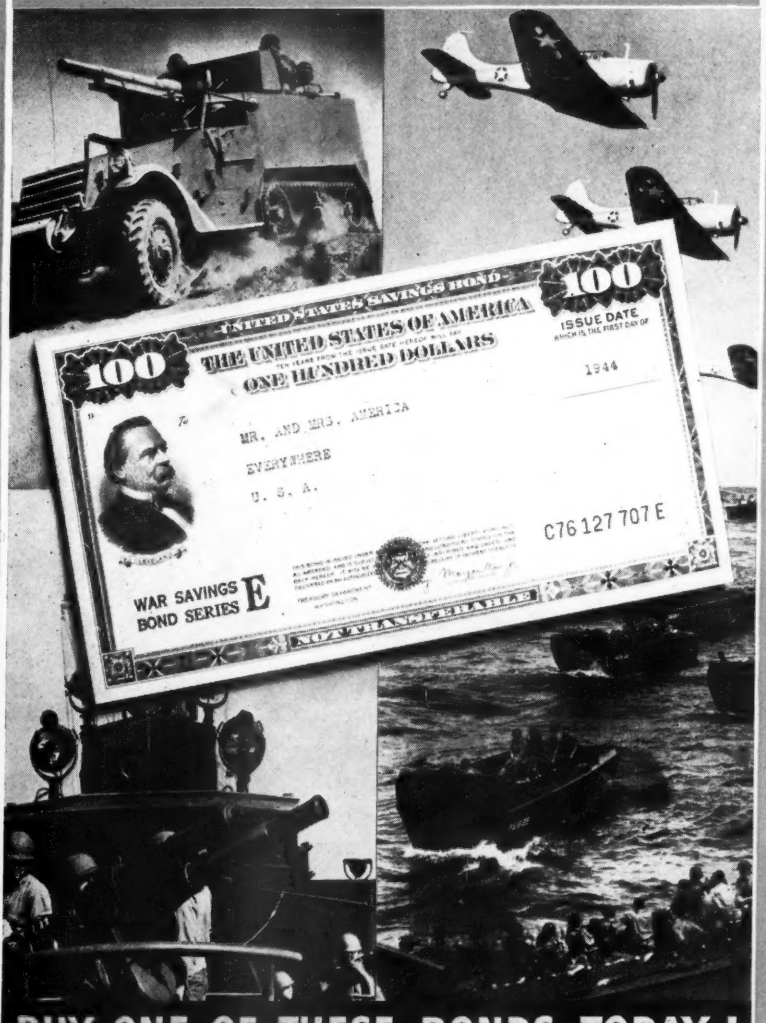
VETERINARY MEDICINE

JULY 1944



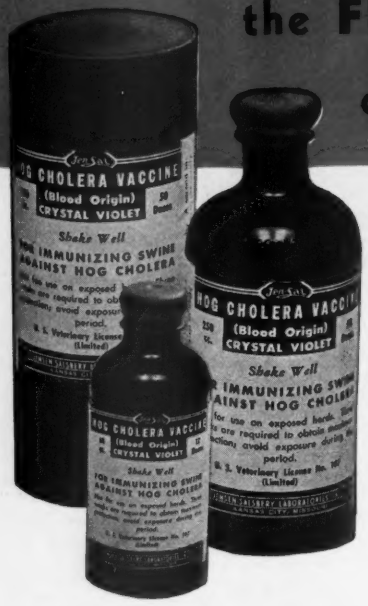
BUY ONE OF THESE BONDS TODAY!

VOL. XXXIX
No. 7

Entered as second-class matter Nov. 8, 1920, at the post office at Chicago, Ill., under the Act of March 3, 1879.

7632 S. Crandon Ave.
Chicago 49, U.S.A.

Cholera Protection without the FIELD HAZARDS of Virulent Virus Usage



AVAILABLE TO QUALIFIED VETERINARIANS ONLY . . . this sterile, nonviable vaccine confers protection of an average eight months' duration when used in a 5 cc. dose on non-exposed healthy swine of all ages—except pigs weaned less than two weeks.

HOG CHOLERA VACCINE

BLOOD ORIGIN

Crystal Violet

Jen-Sal Crystal Violet Vaccine is a carefully standardized and scientifically sound immunizing agent. All lots are safety and potency tested on live pigs—according to B.A.I. specifications—before release for sale.

At once safe and dependable, this new cholera prophylactic, now backed by extensive field usage, opens up new possibilities for veterinary service to the nation's swine industry.

AVAILABLE IN:

Vial 60 cc. (12 doses) Code: VIVAC $2.88

Vial 250 cc. (50 doses) Code: VILET 12.00

JENSEN-SALSBERY LABORATORIES, INC. KANSAS CITY MISSOURI

Write today for an informative booklet and reprints of printed literature.

Kills BACTERIAL INVADERS *in their fox holes!*

WRINKLES, crevices, crypts, and hair follicles are "fox holes" in which bacterial invaders can escape contact with most antiseptics. It takes the *low surface tension* detergent antiseptic PHEMEROL TOPICAL* to penetrate these hiding places and rout Staph and Strep contaminants.

Important in all skin antisepsis, the penetrating potency of PHEMEROL TOPICAL is particularly significant in small animal practice where skin surface conditions are less favorable to effective local therapy.

PHEMEROL TOPICAL is a new type of germicide. It contains no phenol, mercury, or iodine . . . it is a quaternary ammonium compound developed in the Research Laboratories of Parke-Davis. It equals or exceeds tincture of iodine in germicidal action, and is effective in the presence of pus, serum, and sweat.

Available in one-ounce applicator packages, and in one pint and one gallon bottles.

*Trade-mark Reg. U.S. Pat. Off.

PHEMEROL TOPICAL

the *low surface tension* antiseptic

PARKE, DAVIS & COMPANY

DETROIT 32 • MICHIGAN

VETERINARY MEDICINE

JULY CONTENTS 1944

Veterinary Progress, Policies and Comment 263-267

Bond Facts, 263; Paper Packs a War Punch, 263; Paper Conservation, 263; Treatment for Filiarisis, 264; Most Effective Insecticide, 264; Sulfonamide Inhibiting Action of Procaine, 264; Penicillin in Virus Diseases, 264; Bone Cancer Cells of Rats Killed with Penicillin, 264; Penatin Active Against *Brucello abortus* and Other Viruses, 264; Penicillin Dressing, 264; The Surgeon General of the Army at the Medical Field Service School, 265; Veterinary Officers Sent Overseas, 266; Conservation of Leather, 267; Uremia in Baby Pigs, 267; Selection of Swine for Breeding, 267; Equine Encephalomyelitis in 1943, 267; Swine Require Salt, 267.

Demodectic Mange of the Milk Goat 268

Acetonemia—a Vitamin A Deficiency—II 271

Gangrenous Mastitis in Dairy Cows 279

Control Bovine Brucellosis 285

Role of the Veterinarian in Poultry Diseases 287

Clinical Reports 290-293

Mushroom Poisoning in Cattle, 290; Atypical Bladder Calculi of a Bitch, 290; Comminuted Fracture of the Mandible in a Dog, 291; Choosing a Location for Practice, 292; Dog Survives 24 Days' Fast, 293.

Book Reviews 294

Tabers Cyclopedic Medical Dictionary, 294; Virus Diseases in Man, Animal and Plant, 294; What Dog Is That, 294; The New Chemical Formulary VI, 294.

For the Veterinarian

PHARMACEUTICALS

Specialized formulae compounded especially for the needs of the busy practitioner.

BIOLOGICS

Large stock of well known products.

INSTRUMENTS

Complete line of all Veterinary instruments and sundries.

Detroit Veterinary Supply Co.

1792-98 Wabash Ave. Detroit, Michigan

Canadian Branch—Windsor, Ont.

VETERINARY MEDICINE

Registered in U. S. Patent Office

VOL. XXXIX JULY, 1944 No. 7

Published Monthly by the
VETERINARY MAGAZINE CORPORATION
7632 S. CRANDON AVE., CHICAGO, ILL., U. S. A.

$4.00 a year; per copy 50 cents; Special Issues $1.00

All matter appearing in this publication is copyrighted. No part of it may be reproduced without specific permission. Permission will be given if the purpose is unobjectionable. If there be any question as to the propriety of reproduction the author will be consulted.

Entered as second-class matter Nov. 8, 1920, at the post office at Chicago, Ill., under the Act of March 3, 1879. Entered as second-class matter under the title of American Journal of Veterinary Medicine July 19, 1910, at Chicago, Ill., under the Act of March 3, 1879.

D. M. CAMPBELL, *Editor*
7632 S. Crandon Ave., Chicago, 49, Ill.
Telephone South Shore 7160

H. J. MOSTYN, *Eastern Representative*
1265 Broadway, New York, 1, N. Y.
Telephone Murray Hill 4-4037

COPYRIGHTED, 1944, BY VETERINARY MAGAZINE CORPORATION

Bond Facts

More than 55,000,000 Americans own at least one War Bond. That's an average of better than 1½ bond owners for every U. S. family.

Sales of $25 Bonds alone have jumped to 346 million pieces. That's 69 times the number of dollar bills now in circulation.

Of every $100 Americans saved in 1943, $44 was loaned to the government to help pay for the war.

★ AVOID USELESS WRAPPINGS! ★ ACCEPT SIMPLER PACKAGING! ★
PAPER
PACKS A WAR
PUNCH!
FIGHT WASTE!
★ SHARE THE PRINTED WORD! ★ MAKE EACH PIECE STRETCH! ★

The above advertisement, contributed to the war effort by this magazine, was prepared by the War Advertising Council in cooperation with the War Production Board and the Office of War Information.

Paper Conservation

There is an acute shortage of paper. The Government has required users of large quantities of paper to reduce their consumption 23%. It urges those who use lesser quantities to conserve it wherever practicable. For many months we have used lighter paper and, as a further contribution to this effort, the number of pages in this magazine has been materially reduced—beginning with this issue.

By decreasing slightly, spaces between the lines and reducing margins, the number of words on the pages has been increased more than sufficient to make up for the pages eliminated (or will when articles already in type before the change was made have been used); hence, the magazine with fewer pages will contain slightly more reading matter than heretofore.

There is considerable sacrifice in appearance, of course, but it is willingly accepted in view of the present circumstances. The change is not permanent. When the present stringency in the paper supply is relieved VETERINARY MEDICINE will again have the most attractive format in the veterinary field.

The government's appeal to individuals to aid in the paper conservation and salvage program is urged upon our readers. (1) Make each piece of writing paper do its full duty; stretch every inch of wrapping paper. (2) Help your local stores save wrapping paper by discouraging double and extra wrapping of your purchases and carry a market basket or bag to save paper bags. (3) Save every bit of used paper for your local salvage drive. *Paper is a vital need of the War effort. It's ammunition for victory. Think twice before you crumple a piece of it!*

Treatment for Filiarisis

Neosam (stibamine glucoside) a pentavalent antimony compound, used in the treatment of kala-azar has given promise, on preliminary investigation, of being of value in the treatment of filiarisis which a few of our soldiers have contracted in the tropics. Neosam is related to fuadin used in the treatment of heartworm in dogs. There is some prospect that it may be useful in that disease and not require such prolonged treatment as does fuadin.

Most Effective Insecticide

After the war DDT, a powerful new insecticide, will be available for use against the housefly. What it may accomplish in improved sanitation in kitchens, public eating houses, roadside stands, milk processing plants, milk houses and dairy barns is difficult to exaggerate. Flies died within an hour when put into test cages that had been sprayed with the insecticide five months previously, in an experiment conducted by the Bureau of Entomology of the U. S. Department of Agriculture. In a dairy barn, the walls of which were sprayed with DDT, the flies were reduced 95% and remained at a low level for four months.

Procaine Inhibits Sulfonamides

Peterson and Finland[1] point out that the methods for determining sulfanilamide depend on diazotization and the coupling of the resulting diazo compound to produce an azo dye which can be easily estimated by colorimetric comparison. Procaine hydrochloride employed in local anesthesia has a free aryl amine which may enter into this reaction and may inhibit the action of sulfonamides in the blood.[2] The authors find that procaine hydrochloride in amounts ordinarily employed for local anesthesia may be absorbed into the circulation in sufficient concentration to exert a definite inhibiting effect on the sulfonamides that may be present in the blood. Infection introduced into an area which has been infiltrated with procaine may become established locally in spite of the continuous presence in the body of bacteriostatic concentrations of sulfonamide drugs. It is desirable to use local anesthetic drugs other than para-aminobenzoic acid derivatives for infiltration when performing exploratory punctures of potentially infected areas. Procaine and other anesthetics of the para-aminobenzoic acid series should also be avoided in extensive operative procedures on patients having severe infections in which rapid and effective action of sulfonamide drugs is essential.

[1] Peterson, O. L., and Finland, M., *Am. J. Med. Sci.*, 207, p. 166, 1944.
[2] *Vet. Med.*, 37:1, p. 1, 1942.

Penicillin in Virus Diseases

Eighty mice were inocculated with the virus of ornithiosis (psittacosis). Forty were given penicillin; 40 were untreated. Of the treated group, 35 lived, of the controls 35 died.

In test tube experiments at the Wistar Institute of Anatomy and Biology, it was discovered that penicillin will kill rat bone cancer cells while leaving unharmed normal cells growing beside them.

Although both penicillin and penatin are derivatives of the mold *Penicillium notatum*, their action against brucella organisms is by no means identical. While the former has but weak action against *Brucella abortus*, growth is strongly inhibited or completely suppressed by the latter when optimal cultural conditions are used. Penatin is also active against *Escherichia coli* and *Staphylococcus aureus*, whereas penicillin fails materially to modify the growth of these two organisms.—*J. Bact.*

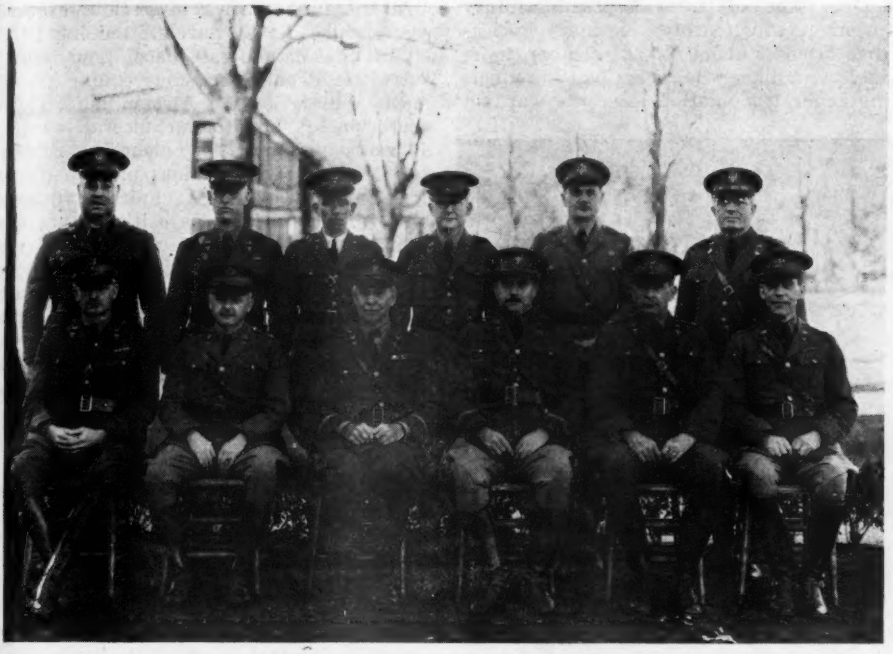
Penicillin Dressing

That glycerine may have a useful role in providing a new type of penicillin dressing is apparent in a recent report* by G. H. Robinson and J. A. Wallace. Knowing that pure penicillin is not available for general use, but realizing that the topical application of a less purified penicillin might be of value in the local treatment of wounds, furnuculosis, sinus infections, gonorrhea and other infections of the skin and mucous membranes, they sought a means to grow the potent drug in direct contact with the lesions. This was finally achieved as follows: A dressing 5 cm. x 5 cm. composed of eight layers of gauze was placed in a Petri dish and saturated with a medium containing 1% yeast extract, 2% dextrose, 2% corn starch and 2% glycerine. This was autoclaved and inoculated with Penicillium, the mold that produces penicillin.

Tests showed that a fair amount of penicillin is produced in the dressing over a period of 4 or 5 days and that a bacteriostatic condition would be maintained at the point of contact with the lesion. Clinical application of the penicillium inoculated dressings on a series of patients who had not been benefitted by other forms of therapy showed the efficacy of the procedure. Although only a few patients were so treated, the evidence thus far obtained offers promise of being a useful addition to the medical armamentarium for treating acute and chronic pyogenic infections.—Glycerine Facts.

*Science, 98:329, 1943.

The Surgeon General at the M. F. S. S.



An illustration of the graduating class, Advanced Course, Medical Field Service School, Carlisle Barracks, Pa., Dec. 15, 1931. The present Surgeon General of the Army (third from left standing) was a member of this class. Those shown in the halftone are: Standing left to right, Major Bertram F. Duckwell; Major Charles C. Hillman (Now Brigadier General, Chief Professional Services in the office of the Surgeon General); Major Norman T. Kirk (Now Major General, The Surgeon General); Major Guy L. Qualls, Major Robert H. Duenner and Major Earl L. Parmenter. Seated, left to right, Major John M. Willis (Now Brigadier General); Major Paul W. Gibson, Assistant Commandant; Brigadier General Edward L. Munson, Commandant of the School; Lt. Col. Delwin M. Campbell, class president; Major Henry P. Carter; Major Herbert W. Taylor, Senior Dental Instructor. All are Regular Army medical officers except Col. Campbell, a veterinary officer, and Major Taylor, a dental officer

The Medical Department of the Army is making a most enviable record in this war even when compared with its brilliant record in World War I.

In the former war 15% of the battle casualties evacuated to Army hospitals died; in this war only 3% die. In 1917-18, deaths from disease were 14.1 per thousand soldiers per year. In this war the mortality from disease is 0.6 man per thousand, per year. In 1943, 173,527 sick and injured were evacuated by air and only 11, or an average of 1 in 15,775 patients, died in transit. The total distance the wounded were evacuated by air in the Mediterranean campaign was 16,491,266 miles and there was only one patient died enroute. In the Tunisian battle, casualties were evacuated to a base hospital 230 miles distant in one hour and 20 minutes. The same trip by motor ambulance required 12 to 15 hours and by hospital train 22 hours.

The veterinary service, which is a part of the medical department of the Army, has exacting tasks of great magnitude in this war. Food must be inspected in quantities never before envisioned. This food is procured, not only from the usual sources but much of it from processors new in this field and unfamiliar with the manufacture, storage and packaging of food products, which increases greatly the difficulty of supervising the processing and inspecting the finished product. The great quantities of dehydrated food procured for overseas forces has entailed many new and complicated problems of inspection. The enormous demand for fluid milk by the armed forces in this country has necessitated the procurement of milk from thousands of dairy farms and hunreds of processors whose standards of sanitation did not meet Army requirements. This made it necessary for the veterinary service to engage in extensive educational and "clean-up" campaigns to qualify these sources. The same situation prevailed

also in creameries, cheese factories and processing establishments for other dairy products. One result of the widespread sanitary improvement which army veterinary inspection has brought about will be cleaner, more palatable and more wholesome dairy products for the general population after the war.

Ten of the Regular Army senior veterinary officers were ordered to Africa, Italy, India, China and Southeast Asia a few months ago. Five of them are shown here on their arrival somewhere in India. From left to right they are: standing, Col. Jack Fuller and Col. Harry E. Van Tuyl; sitting, Col. Daniel Mallan, Col. Isaac O. Gladdish and Col. Raymond I. Lovell. All have seen service in the Philippine Islands and thus have had first-hand experiences with tropical diseases

Veterinary officers and veterinary enlisted personnel on foreign duty are confronted with many new and difficult tasks. The animals of the "old army" included only horses and mules and their diseases comprised, in the main, only those prevalent in this country with which of course all veterinarians are familiar. But now the list of animals includes also War Dogs, African burros, Chinese ponies, Indian bullocks, etc.—almost any animal that can draw a cart or carry a pack. Since in this war all local food supplies available are drawn upon for the subsistence of our overseas forces the diseases of food animals, all over the world, concern our army veterinary service. The problem is extremely complicated in India, China, Burma and the Islands of the Western Pacific; Asia being the original home of most of the plagues and the present habitat of almost every disease to which animal flesh is heir.

All those more or less mysterious, exotic diseases, which, for us, have existed only in textbooks—mal de caderas (baccy poy), murrina, surra (el debab), nagana, equine piroplasmosis (biliary fever), African horse sickness (perdeziekte), blue tongue, ulcerative cellulitis epizootic lymphangitis (culebrilla), rinderpest (cattle plague), contagious pleuropneumonia, foot-and-mouth disease, canine piroplasmosis and various tropical parasitisms—are everyday experiences to our overseas veterinary service; while glanders, anthrax, dourine (maladie du coit), vesicular stomatitis, mange, myaises, purpura hemorrhagica, trichophytosis and others more or less uncommon here, are ever present at some of the world-wide stations of our military forces.

Is Your Convention Justified?

"Although a number of organizations, large and small, have canceled conventions in compliance with the request of the Government, many others are continuing to hold 'conventions as usual,'" says Col. J. Monroe Johnson, director of the Office of Defense Transportation, in a release from the O.D.T. late in May.

"We are obliged to report that even with the prospect of invasion and the resulting need to keep transportation lines clear, unnecessary trade shows and conventions continue to be planned. The governing officials of these organizations obviously have neglected to take into consideration the harm these conventions inflict on war-transportation efficiency.

"The O.D.T. has repeatedly requested that conventions and other travel-stimulating meetings be canceled for the duration. Convention travel is especially disruptive because of the concentrated impact of its additional passenger volume on the war-limited transportation facilities. The resulting traffic congestion is far beyond what might be expected from the small percentage of passengers that conventions add to the total number carried.

"Holding unnecessary conferences in wartime is outraging sensibilities of business men. They feel that they are being badgered into attendance at a time when convention-going is impeding essential travel, and when they would, by preference, stick strictly to the war job at home.

"Organized troop movement and casualty transportation is gaining record-breaking proportions. The hardships involved in the cancellation of conventions involving travel cannot be compared with outright danger such non-essential travel imposes on the war effort."

To conserve leather, the Army Quartermaster has developed a harness made of cotton webbing. The material is chemically treated against moisture, mildew and weathering and is of strength equal to leather harness. With care it is expected the new type harness will be serviceable for ten years.

Uremia in Baby Pigs

Heavy death losses of pigs during the first two weeks of life have occurred recently in the Beltsville herd. A high percentage of these appear to have had uremia, a disease in which excretory products that are normally given off by the kidneys accumulate in the blood and finally cause death. The condition is characterized by deposits, presumably urates, in the uriniferous tubules (excretory ducts of the kidneys) and a marked increase in urea, uric acid, and other compounds in the blood, kidneys and liver. The blood-sugar content of most of the affected pigs is within the normal range, a fact that appears to differentiate this disease from the so-called baby-pig disease due to hypoglycemia (low blood sugar). The cause of uremia is not known and success has not been attained in treating the disease.—Ann. Rept., B.A.I., U. S. Dept. Agr., 1943.

Selection of Swine for Breeding

As a result of experiments at the Regional Swine Breeding Research Laboratory at Ames and the study of swine breeding records at state experiment stations in the Corn Belt, Dickerson and Hazel have developed a new swine breeding guide that they believe will speed up herd improvement.

Genetic improvement is computed in the two major factors of successful swine production—the growth rate of pigs and the productivity of sows measured by the number and weight of litters at weaning time. Their conclusion is that heredity is responsible for 30% of the variation in the growth rate of pigs to the age of six months, 15% of the variation in the weaning weight of pigs and 18% of the productivity of sows.

The authors' plan provides for culling (castrating) two-thirds of the boars at weaning and retaining the remainder till they are five to six months of age, at which time 80 to 90% of them should be castrated.

About twice the number of gilts that are to be retained for breeding should be kept in the breeding herd until they reach the age of five to six months and then the culling should be completed. However, even more rapid progress can be made by postponing final culling of the sows until they have weaned their second litter.

The laying house mortality rate for poultry declined from 19.4% for 1942 to 16.2% for 1943. This effected a saving of 70 million pounds of poultry meat.

Equine Encephalomyelitis in 1943

The incidence of encephalomyelitis in the United States was low last year—only 4,768 cases being reported to the federal Bureau of Animal Industry. The deaths numbered 1,622. As in several preceding years the outbreak centered in Iowa which reported 675 cases. Minnesota, 423 cases, Nebraska, 398 cases, Michigan, 367 cases, Illinois, 272 cases, Oklahoma, 285 cases and Indiana, 161 cases, included more than half of all the cases. However, 760 cases were reported from California and all the Mountain and West Coast states experienced many cases. There were very few cases east of Indiana or south of the Ohio River.

Michigan horses where the eastern type of the disease prevailed again this year for the second time, suffered the greatest mortality. The deaths reported numbered 349 but the official reports were incomplete. Michigan veterinarians have stated there were no recoveries.

Swine Require Salt

Owing to the shortage of animal protein, hogs are being fed less tankage than formerly and more soybean, peanut, linseed or other oil meals. When this is the case it should not be overlooked that the animals require more salt.

Salt is not just a condiment, added to the animal ration to make it more palatable, but an essential mineral. Swine and other domestic animals become unthrifty when given insufficient salt regardless of how nutritious the remainder of the ration may be. Even when the ration of swine was balanced by tankage, which is rich in salt, one of the Canadian agricultural experiment stations determined that supplying additional salt increased the gain from the feed consumed; one pound of salt, in effect, saving seven pounds of grain.

At the Iowa experiment station, where a swine ration was balanced with oil meal instead of tankage, one pound of salt saved as much as 213 pounds of grain as compared to control groups which were fed a ration without any additional salt, and the other received it *ad lib*.

Professor Bohstedt of the University of Wisconsin recommends that hogs be supplied in their ration one-half pound of salt for each 100 pounds of grain consumed and, in addition, that it be made available, "free choice."

Demodectic Mange of the Milk Goat*

By A. J. DURANT**
Columbia, Missouri

DEMODECTIC mange in the milk goat has been observed in a number of herds in Missouri during the past five years, thus furnishing an opportunity to make observations and to study this parasitic disease. The lesions in a number of cases were observed and letters had been received describing other cases, before discovering the demodectic mange mite in the nodules of the infected animals.

History

Cram,[1] 1925, made the first report of the disease, followed by a report[2] from the Division of Experimental Surgery and Pathology, Mayo Foundation, the same year. It appears from these papers that both reports refer to the same herd. In both instances the goats were obtained from a purebred Toggenburg herd in St. Louis. Cram gives a rather complete review of the foreign literature, with photographs and drawings of the mites. She states that her report is "what is believed to be the first report of demodectic mange of the goat as having occurred in the United States."

It should be mentioned that at the same time Doctor Cram received the specimens at Washington, the Veterinary Division of Iowa State College received material from the same case and reported the demodectic mange mite as being found and that the condition is considered incurable.

It is peculiar that all of the following cases observed were in Toggenburg goats.

Cases

No. 1. The first case observed in Missouri was a purebred Toggenburg milking doe. She had just been clipped and about 60 nodules were present on her sides, shoulder, neck and jaws. At that time demodectic mange was not suspected and further investigations were not made.

No. 2. Later an affected doe was found in a herd of approximately fifteen head of Toggenburgs in a central Missouri city, about 25 miles from the first case. This doe was retained in the herd about one year after the disease was observed. She was not treated, yet there was no spread of the disease to the other goats.

No. 3. In another herd of 25 purebred Toggenburgs in north central Missouri, one animal was found to be infected; however, the remaining 24 goats remained healthy.

No. 4. The fourth case occurred in central Missouri in a herd which consisted of only three goats, two of them Alpines and the other a grade Toggenburg wether. The Toggenburg was the only goat affected.

No. 5. The fifth case was from Connecticut. The illustrations for this paper were taken of an animal from this purebred Toggenburg herd.

Other cases previous to these had been reported by mail, so it might be said that demodectic mange in goats is widespread in the United States, though often not recognized as such. This is in accordance with the report from the Mayo Foundation which said that "the disease is probably rare, especially in the United States. *However, it may be that its recognition is more rare than its occurrence.*"

Fig. 1. Head and neck of an adult Toggenburg milking doe affected with demodectic mange. The clipped area shows the nodules. The cross lines illustrate the position of incisions made in treatment of each nodule

This is likely the crux of the matter. There are several specific reasons why such may be the case: (1) the disease is not fatal, (2) it does not take epidemic form and (3) the comparatively long hair of the Toggenburg goat hides the lesions and they may remain undetected for a considerable period.

The photograph of the mange mites (Fig. 3) was obtained from one of the lesions, or nodules, from the herd in Connecticut. Other specimens were taken from some of the cases observed in central Missouri.

In one of the herds (No. 2) in central Mis-

*Journal Series No. 945, Department of Veterinary Science, Missouri Agricultural Expt. Station.

**Veterinary Department, University of Missouri.

[1] Cram, Eloise B., Demodectic mange of the goat in the United States. *J.A.V.M.A.*, 66:4, pp. 475-480, 1925.

[2] Hardenbergh, John G., and Schlotthauer, Carl F., Demodectic manage of the goat and its treatment. *J.A.V.M.A.*, 67:4, pp. 486-489, 1925.

souri, the owner had a Chow dog affected with mange for several years and, naturally, suspected that the mange in the goat may have been contracted from the dog. This probably was not the case for it is unlikely that demodectic mange of the goat would be contracted from affected dogs. In most cases it is difficult to transmit this form of mange from one dog to another.

Fig. 2. Clipped area on the body of the goat shown in Fig. 1. This doe had a total of 88 nodules on the head, neck and body

Microscopic Examination of Nodules

As soon as it was suspected that these nodules in the milk goat might be caused by the demodectic mange mite specimens were immediately obtained. The nodules were incised by opening the skin directly over the nodule and removing a grayish-white, puslike, semi-cheesy material. When examined under the microscope the material was found to be a mass of demodectic mange mites, detritis, and other excretory products which usually accompany such material. The findings corresponded to those described by the authors mentioned previously.

The accompanying photographs of some of the mites are characteristic of those found in all cases. Following the suggestions of Cram, this parasite is designated as *Demodex folliculorum* var. *caprae*, assuming that it is the same as reported previously. The lesions on the skin, as illustrated by the accompanying illustrations, will enable veterinarians to diagnose this disease without a microscope. In every case possible, however, the clinical diagnosis should be confirmed by microscopic examination.

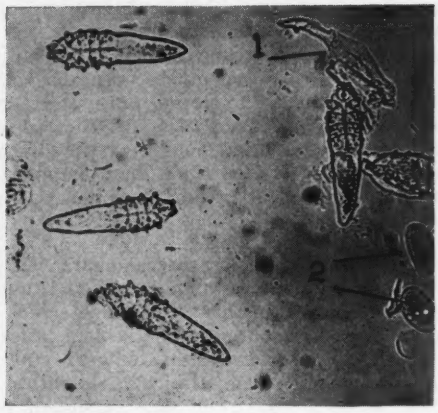


Fig. 3. Photomicrograph of *Demodex folliculorum* var. *caprae*, showing adults, a skin cast of the parasite (1) and eggs (2). Magnified 100 diameter

Symptoms

In some cases observed no symptoms of dermatitis, such as scratching, were noted. In other cases the owners reported that the animals scratched or rubbed the affected parts against objects, indicating a definite itching. In some animals the hair was rubbed off, especially on the sides of the jaws. In most of the cases observation of the goat alone would not have revealed the presence of the lesions caused by the parasites. In other cases the presence of the nodules were evident under the hair on the side of the jaws and could be felt by running the hand lightly over the skin. However, after a goat was clipped the nodules were clearly evident.

In one animal, 83 nodules were present. In another herd the number and location of the nodules were as follows: front legs from knee up, 8; head and jaws (none on top), 10; neck, 25; chest, 3; shoulders, 20; sides, 28; and rump, 2. The total number was 96. Forty to fifty nodules in the skin around the neck, shoulders, sides and back have been reported.[2] It would appear from observation of other cases that the number of nodules usually found in a well developed case is between 80 and 98, since the nodules of the two cases reported were carefully counted. The size of these nodules as observed corresponded very closely to those described[1,2] earlier. The nodules observed varied in size from .5cm to 1.25cm in

diameter. One case (No. 4) should be mentioned, however, in which the diameter of the nodules was very much larger than the largest reported. In this case the owner had attempted to treat the lesions by making an incision in the skin and squeezing out the contents of the nodule. Because medicinal treatment did not follow the expulsion of the pus, the nodule immediately began to enlarge. In practically all improperly treated nodules there was great enlargement. The nodules varied in size up to 5cm. They were raised much more above the surface than the unmolested ones, and their surfaces were denuded of hair. This is not regarded as the usual course of the disease but as an illustration what may happen if the nodules are not properly treated.

Spread of the Disease

After observation for several years, it appears that demodectic mange, although quite common and in many cases undetected in herds of Toggenburg goats, seldom affects more than one or two individuals in a herd. Also the spread is almost negligible within a period of two years. The slow spread of the disease may be explained by, (1) the parasites do not, as in the dog, infect the hair follicle, (2) they may enter the goat's skin only through the openings into the sebaceous, or other glands of the skin, and for that reason a goat might be exposed a number of times without ever contracting the disease. In one herd (No. 2) of about 15 goats, in which only one case of demodectic mange was found, another adult Toggenburg developed a scaly, scurfy condition of the skin in the posterior fetlock region of all four feet. Since its appearance was somewhat similar to red mange in the dog, scrapings were taken from the area, but microscopic examination failed to demonstrate the *Demodex folliculorum* var. *caprae*.

Treatment

The infected goat (herd No. 2) was secured for treatment. She was clipped very closely over the entire body with an electric clipping machine. The nodules were easily observed and 83 were counted. The treatment was practically the same as that used in earlier reports.[2]

In the treatment of the case described a cross incision was made immediately through the nodule (Fig.1) with a Bard-Parker scalpel (handle No. 3 and blade No. 12). The cross incisions were made entirely through the skin into the contents of the nodule. Then by means of a curette (Fig. 4), the content of each nodule was removed carefully. A few drops of a 10% carbolic acid solution was then injected into each sac. The entire area was then painted with tincture of iodine. Two weeks after the operation the animal was inspected and only in one or two places could the remnant of a nodule be found. The goat was sold as a healthy animal six months later and has remained well ever since, there being no further evidence of the parasite or its lesions.

When treating infected goats it should be kept in mind, first, that they should be clipped very closely so that the lesions may be more readily detected, and second, the treatment must be very thorough. That is, the incisions through the nodules must be sufficiently deep so that the material can be properly evacuated as described, and then the medicinal treatment should follow.

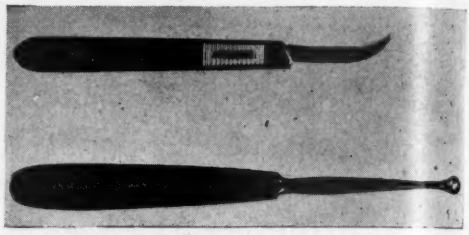


Fig. 4. Knife and curette used in the treatment of the nodules. The contents of each nodule must be completely evacuated

The publication of this report of mange in the milk goat should be of interest to veterinarians who are in areas where the milk goat is present in such numbers as to constitute an industry. It is estimated that there are six million milk goats in the United States and that they produce approximately five million quarts of milk a day. For the past 14 years the author has given considerable time and attention to the study of diseases and parasites of milk goats.

Other Skin Injuries of Goats

Goats are frequently subject to scratches, punctures and infections from thorns, awns, spines and burrs. Thus many plants that are not poisonous to goats may be quite troublesome by causing mechanical injuries with consequent infection. Often a herd will come in from pasture or woodlot with udders badly scratched, or with less noticeable injuries or thorn punctures on other parts of their bodies. Frequently, if one runs an inquiring hand over the goat one can feel thorns or burrs. Often these cause a break in the skin, allow infection to enter, and sometimes lead to abscesses. The rather common occurrence of abscesses in the head and neck of the goat may well trace their inception to such punctures.—*Dairy Goat Bulletin.*

Acetonemia, a Vitamin A Deficiency—II

By JOHN W. PATTON
East Lansing, Michigan

IN a previous discussion[1] it was pointed out that acetonemia is extraordinarily prevalent among the milk cows in the Cotton Belt during January, February, and March when the commonly used feed of cows in that section is extremely low in vitamin A. It was also reported that recovery in cows affected with uncomplicated acetonemia was prompt and complete when vitamin A was given to them and, finally, that primary acetonemia is simply a manifestation of vitamin A deficiency.

In this, as in the preceding discussion, "acetonemia" is used to designate the condition generally called by that name by veterinarians, particularly in the South—a condition accompanied by acute clinical symptoms. "Ketosis" was and is used to designate the condition in cattle in which the blood and urine changes are similar but the clinical symptoms of acetonemia are absent or unimportant. "Ketosis" is employed in medical literature to designate both the mild and the severe type of the ailment in man. The symptoms were described in the preceding article and need not be repeated here.

As explained in the initial report, approximately one-half of the season of greatest prevalence of acetonemia was spent in making a survey of the conditions under which acetonemia occurs in 15 states. The history of the disease indicated that the prevalence could be expected to subside rapidly at the end of March. Thus we were confronted with delaying further study until next winter or to return to East Lansing, assemble the necessary personnel, scientific apparatus and transport it to Mississippi as quickly as possible and continue the study this season. The latter plan was chosen.

However, unforseen delays intervened and it was the 29th of March before the laboratory was set up in Corinth, Miss., and ready for operation. The investigation was hampered greatly by this delay. At the middle of February, four to 10 cases of acetonemia, severe enough to require the services of a veterinarian, were occurring daily in the Corinth section. By the end of March the problem of finding uncomplicated cases was a difficult and time-consuming one. In February 85% of the cases seen were uncomplicated and only 15% complicated by other ailments—chiefly of uterine origin. In April the prevalence of complicated cases was about the same but the uncomplicated cases had largely disappeared with the availability of green vegetation to the animals. As a result a considerable part of the work planned was perforce put off until next season but enough progress was made to justify a further report.

Another difficulty encountered with the first appearance of green leaves on honeysuckle, wild crab, wild plum and wild cherry trees was a considerable amount of plant poisoning. As the owners of the poisoned animal often mistook the symptoms for those of acetonemia, this resulted in many calls to such animals and thus entailed loss of time and effort.

However, it was possible to study a few cases (far fewer than had been planned) from the following aspects:

1. The blood sugar and blood and urine acetone levels in clinically normal cows.

2. Effect of the administration vitamin A on the blood sugar, the blood and urinary acetone levels and the milk production of clinically normal cows.

3. The same factors in cows suffering from acetonemia and ketosis.

4. The vitamin A, carotene, sugar, acetone and hemoglobin content of the blood of clinically normal cows and of cows affected with acetonemia either as a primary or secondary ailment.

In the past 12 years there has been a great deal of research into the cause and nature of acetonemia (ketosis) in cattle. The economic importance of the problem is further attested by a much larger number, who have written upon the subject from clinical or other aspects. The agreement of these various research workers and clinicians on the essential facts in connection with the ailment is truly remarkable. Detailed studies by Alston,[2] Boddie,[3] Boley,[12] Brouwer,[4] Butts,[5] Cutler,[5] Dijkstra,[4] Duell,[5] Duncan,[6] Fincher,[7,8] Hallman,[5] Hayden,[8] Huffman,[6] Knodt,[9] Lormore,[10] Roepke,[11] Sampson,[8,12] Shaw,[9] Sjollema, Tobin,[6] Udall,[13] White[9] and a number of others, have contributed to the knowledge and understanding of the subject.

The consensus regarding acetonemia may be said to have established:

1. That a low level of blood sugar is characteristic of severe cases.

2. That there is a high concentration of acetone bodies (ketones) in the blood and urine.

3. That the majority of the cases occur from December to April—a marked seasonal occurrence.

4. That the ailment usually occurs in high-producing, well-nourished cows from five days

to six weeks or longer following parturition.

5. That a low blood sugar level and high ketosis may occur without clinical symptoms.

6. The more recent research indicates that there is liver impairment or dysfunction.

In addition to the above (except for part of No. 4) our own investigation establishes:

1. Primary, uncomplicated acetonemia (ketosis accompanied by clinical symptoms) is far more prevalent in the Southern States than it is in the central and northern portions of the country and that the same blood and urine chemical picture prevails in the South as elsewhere.

2. That acetonemia ordinarily occurs in high or fairly high-producing cows, which are maintained on a diet inadequate for their requirements in quality or quantity, or both. It is not limited to well-nourished cows but may appear in any bovine animal on a diet that is relatively inadequate. It depends upon the relation between the intake and output. The higher producers in a herd may suffer on a diet that is adequate for the average cow in the herd. Further it occurs frequently as early as the second day after parturition and sometimes, though rarely, earlier.

3. That depletion of the vitamin A reserve of the animal by the colostrum and flush milk production is indicative of the impaired liver function, the altered blood and urine chemistry and the clinical symptoms which occur in the usual cases of primary, uncomplicated acetonemia, i.e., this type of acetonemia is a vitamin A deficiency. Further, marginal vitamin A deficiency may be a contributing factor in acetonemia which occurs as a complication of metritis and other debilitating ailments.

Theories as to the Cause of Acetonemia

In acetonemia there are two important departures from the normal chemistry of the blood—the sugar is lowered and the ketone bodies are raised. Various theories as to the cause of the ailment have been built around these facts. Briefly stated, the ones of widest acceptance have been:

1. The carbohydrate intake is insufficient, with a resulting increased fat metabolism.

2. The glycogen stored in the liver is not released to the blood or the carbohydrates in the diet are not utilized to a normal degree and as a consequence the metabolism of fat is raised.

3. Because sufficient carbohydrates are not available, increasing quantities of fats are used but not completely oxidized.

4. The increased ketones in the blood may be due to incomplete oxidation of fats with the production of toxic oxidation products. In all of these theories the liver is implicated.

Our theory is that primary acetonemia is due to an impaired function of the liver which often results from a lack of vitamin A. Support of this is to be seen in (1) the rapid and complete disappearance of the clinical symptoms, (2) marked rise in the blood sugar level, (3) almost immediate decrease of the amount of ketone in the blood to normal and (4) the promptness with which milk production returns to normal and often above that produced before the attack; when large doses of vitamin A are administered.

The foregoing is based upon the observations reported in the preliminary discussion, to which reference has already been made, and to the subsequent observations reported here.

Methods Employed

When a case of acetonemia was reported the animal was seen and a diagnosis was made by Dr. W. L. Stroup or the writer. In most cases, a sample of blood was then drawn from the jugular vein and a sample of urine was obtained, followed by the administration in capsule of the selected dose of vitamin A.

Most of these cows were seen daily during the critical stages. If conditions indicated that it was desirable, blood and urine samples were obtained later and the analyses compared with those of sample obtained prior to treatment.

Vitamin A and carotene determinations were made by a modification of the Dann and Evelyn method (*J. Biochem.*, 32, p. 1008. 1938).

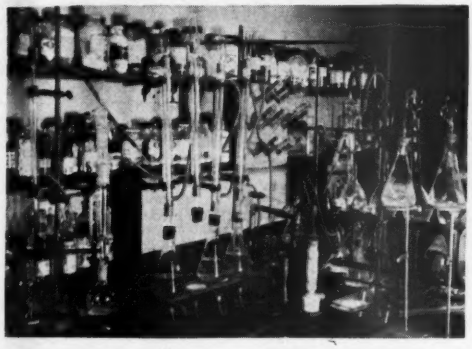
The blood sugar was determined on Somogyi filtrate (1930). Since blood sugar values are lowered by digestion of glucose on standing, efforts were made to obtain the filtrate as quickly as possible. In most cases, proteins were precipitated out of the blood within about 30 minues of the time the sample was drawn from the cow.

During the early part of the study blood

TABLE I—SUMMARY OF VITAMIN A ANALYSES OF BLOOD PLASMA

	Before Treatment		After Treatment	
	Carotene I.U./100cc	Vitamin A I.U./100cc	Carotene I.U./100cc	Vitamin A I.U./100cc
Cow 1	1330.	138.	355.	140.
Cow 3	1580.	88.	...	...
Cow 4	1500.	12.	...	...
Cow 6	1035.	4.	996.	80.
Cow 7	1852.	29.	...	...
Pooled normal	382.	81.	302.	204.

sugar was determined on the Somogyi filtrate by the Hoffman method (1937) using a Cenco photelometer calibrated with CP glucose. It was observed that the blood sugar values tended to run somewhat higher than those reported in the literature for cow's blood. Also, it was obvious that the Hoffman method was not ideal for measuring low levels of blood glucose because the range of the readings was relatively small. For these reasons, later determinations were made by the Folin modification of the Folin-Wu method. As is shown by Table I, fair agreement was obtained by the two methods applied on the same filtrate.



A laboratory was set up in Corinth March 29th and dismantled April 8, 1944

Urinary acetone bodies were determined as acetone by the method of Van Slyke (1917) using a sample of filtrate equivalent to 2.5cc of urine, the milligrams of precipitate were multiplied by 2.48 to obtain milligrams of acetone bodies calculated as acetone per 100cc of urine.

The blood acetone bodies were determined by taking 10cc of whole blood with 100cc of water and adding 20cc of cold 10% mercuric sulfate solution. The mixture was shaken, made up to 250cc and, after 15 minutes filtered. 125cc of this filtrate was treated as in the method for determination of acetone bodies in the urine. The milligrams precipitate were multiplied by 1.24 to give the milligrams of total acetone bodies as acetone per 100cc of blood. The objections to this procedure have been reviewed by Barnes and Wick (1939) but the method is believed to give results that are satisfactory from a comparative standpoint.

Milk samples were examined using the procedure followed for blood samples.

Hemoglobin was determined by the method of Sheard and Sanford (1929).

A number of urine samples were examined for albumin, using the nitric acid ring test as described by Hawk and Bergeim (1927).

TABLE II—COMPARISON OF HOFFMAN AND FOLIN BLOOD SUGAR METHODS

	Milligrams Glucose per 100cc	
Sample	Hoffman Method	Folin Method
C-11	22.5	21.0
C-13	57.5	59.5
C-14	56.0	58.8
C-15	46.5	51.0
C-16	56.0	59.0

Case Records

C-1. Jersey Cow.—First seen March 29, 1944. This cow was sick on the morning of March 29th and had been given an ounce of chloral hydrate at that time. At three o'clock that afternoon the vitamin A treatment was commenced. Her calf was two weeks of age. The cow and the calf had been purchased ten days previously by the present owner. Her diet prior to purchase is unknown. For the past ten days she had been fed cottonseed meal and hulls and lespedeza hay.

Symptoms: Staggering. Needed help to rise. Breath characteristic. Constipated.

Treatment: 500,000 USP units vitamin A.

March 30: Apparently normal but not eating well. Metritis was suspected.

March 31: Uterine discharge present. Condition otherwise normal. Eating. Given 1,000,000 units of vitamin A and 25mg diethylstilbestrol.

April 1: Condition normal. Given 1,000,000 units vitamin A.

April 3: Condition normal. Given 1,000,000 units vitamin A.

Laboratory Data: See Table VI.

C-2. Jersey Cow. First seen March 30, 1944. The ration consisted of cottonseed meal and hulls (about 21 pounds per day) and 1½ gallons shelled white corn daily. Peanut vine and soybean hay. Her calf was six weeks of age.

Symptoms: Staggering, falls down. Head high. Characteristic breath. Ross test: 1 plus.

The owner reported that milk production had fallen from three gallons per day to one quart at the evening milking on March 29th.

Treatment: Given 1,000,000 USP units of A.

March 31: Appearance normal. Eating. Given 1,000,000 units vitamin A.

April 1: Normal. Eating. Milk production increased to three quarts morning and evening. Given 1,000,000 units vitamin A.

April 6: Normal. Milk production reported to

TABLE III—EFFECT OF ACETONEMIA ON COMPOSITION OF COW'S BLOOD AND URINE

	Blood Vitamin A as I.U./100cc plasma		Blood Sugar	Acetone Bodies as mgms% Acetone			Hemoglobin Urinary	
	Carotene	Vitamin A	Mgms%	Blood	Urine	Milk	Gms/100cc	Albumin
Normal*	676.	71.	56.9	2.0	37.	...	10.4	...
Acetonemia* .	1035.	4.	34.1	43.7	274.	52.0	10.8	...

*These data were selected and do not represent all observations made, but are believed to be typical for the cases seen.

be greater than when the cow first freshened.
Laboratory data: See Table IV.

C-3. Jersey Heifer.—First seen March 30, 1944. A very small heifer, weighing only about 400 pounds, 16 months of age. Emaciated. Fed cottonseed meal, white corn, a little hay of poor quality. Dystocia the preceding night. Calf six hours of age.

Symptoms: Very weak, unable to stand when lifted to her feet. Ross test: negative. It may be noted in Table IV that, notwithstanding the negative Ross test, the blood and urine acetone content was high.

Treatment: Given 1,000,000 USP units vitamin A.

April 1: Put in a sling. Seemed much improved but still very weak. Given 1,000,000 units of A.

April 6: Improving. Able to get about normally. Suckling calf. Extremely thin. Good appetite. Given 1,000,000 units vitamin A.
Laboratory data: See Table IV.

C-4. Hereford Cow.—First seen March 31, 1944. Had received little or no grass. Ross test: 1 plus. No marked symptoms.
Laboratory data: See Table VII.

C-5. Hereford Bull.—Same feed as C-4.

TABLE IV—BLOOD AND URINE VALUES OF TWO UNCOMPLICATED CASES OF ACETONEMIA TREATED WITH VITAMIN A.

Cow No.	Date	Blood Sugar Mg%	Blood Acet. Mg%	Hemogl. Gm%	Urine Acet. Mg%	Treatment USP Vit. A*
C-2	3/30	27.5	...	...	116.0	1 Million
	3/31	37.0	64.6	...	126.0	1 Million
	4/1	...	...	...	136.0	1 Million
	4/6	54.5	2.0	8.6	16.0	None
C-3	3/30	17.0	73.0	...	227.0	1 Million
	4/1	44.0	trace	....	....	1 Million
	4/6	68.0	2.5	11.3	....	1 Million

*Fish liver oil, 1 million USP units Vitamin A per gram.

TABLE V—BLOOD AND URINE VALUES OF FOUR NORMAL* COWS BEFORE AND AFTER VITAMIN A THERAPY.†

Cow No.		Blood Sugar milligrams percent	Blood Acetone milligrams percent	Blood Hemogl. grams per 100cc	Blood Vit. A** I.U. per 100cc	Blood Carot.** I.U. per 100cc	Urine Acetone milligrams percent
C-12	b	58.5	3.0	10.9	81	382	25.0
	a	66.5	8.0	11.1	204	302	24.0
C-13	b	57.4	Trace	7.8	81	382	...
	a	70.8	1.6	10.6	204	302	...
C-14	b	48.7	Trace	10.5	81	382	30.0
	a	48.0	1.1	13.2	204	302	3.0
C-15	b	57.5	7.8	11.5	81	382	83.0
	a	66.0	Trace	12.2	204	302	4.5
Average	b	55.5	5.4	10.2	81	382	21.1
	a	62.8	3.6	11.8	204	302	10.5

Note.—The elapsed time between first and second analysis was 48 hours.
*Cows without visible symptoms.
†One dose of one million USP Units Vitamin A orally in capsules.
**Vitamin A and carotene determinations were made from pooled blood from all four cows. Hence the quantities given do not apply to any *one* animal as the table appears to indicate.

TABLE VI—METRITIS COMPLICATED WITH ACETONEMIA

Cow No.	Date	Blood Sugar mg%	Blood Acetone mg%	Blood Hemogl.	Urine Acetone mg%	Urine Ross Test	Milk Acetone mg%	Treatment USP units Vitamin A
C-1	3/29	...	345.0	...	627.0	4X	....	500,000*
	3/30	...	...	...	606.0	..	....	1,000,000
	3/31	...	...	...	313.0	..	....	1,000,000†
	4/3	52.7	37.9	14.1	155.0	..	....	.....
	4/5	47.8	25.0	14.3	100.0	..	....	1,000,000
C-6	3/31	58.0	25.8	...	152.0	1X	144.0	1,000,000
	4/1	74.0	...	...	128.0	..	....	1,000,000†
	4/3	70.0	9.4	...	90.0	1X	3.2	1,000,000
	4/6	60.0	23.1	10.3	147.0	..	8.7	1,000,000
	4/7	30.0	25.3	9.4	....	..	....	.....
	4/8	...	26.5	9.8	380.0	1X	....	1,000,000
C-11	4/3	23.8	37.9	...	97.0	2X	....	1,000,000†
	4/5	21.8	35.0	13.7	80.0	2X	....	1,000,000

*Note: One ounce of chloral hydrate was given six hours prior to first treatment with Vitamin A.
†Note: Given 25mg diethylstilbestrol in addition to the Vitamin A.

Laboratory data: See Table VII.

C-6. Jersey Cow.—First seen March 31, 1944. Fed cottonseed meal, cottonseed hulls and dairy feed, also lespedeza hay. Calf two weeks of age.

Symptoms: Those of a mild case of acetonemia, probably with uterine complications. Milk had a pronounced disagreeable odor. Nervous. Characteristic breath. Appetite in abeyance. Ross test: 1 plus.

Treatment: Given 1,000,000 USP units of A.

April 1: Given 1,000,000 units vitamin A and 25mg diethylstilbestrol. Condition somewhat improved. Eating.

April 3: Given 1,000,000 units vitamin A. Condition same, or slightly improved. No odor in milk. No albumin in urine.

April 6: Condition same. 1,000,000 units vitamin A. Animal in estrus.

April 7: Some discharge from uterus. Given 1,000,000 units vitamin A. Still in heat.

April 8: Condition same. Estrus subsiding. Owner reported decreased milk production. Observation discontinued (laboratory closed).

Laboratory data: See Table VI.

C-7. Normal Jersey Cow.—First seen March 31, 1944. Fed a good ration. Calf several months old. Kept on a well managed dairy farm. Had been on relatively good green pasture for two weeks. Ross test: Negative.

Laboratory data: See Table VII.

C-8. Apparently Normal Jersey Cow.—First seen April 1, 1944. Kept in a dry lot with very little pasture. Not giving milk. Given 1,000,000 units vitamin A.

April 3: Given 1,000,000 units vitamin A. Urinary acetone bodies: 13mg%. No albumin in urine.

Laboratory data: See Table VII.

C-9. Apparently Normal Jersey Cow.—First seen April 1, 1944. Same herd and same condition as C-8. Due to have calf in week or ten days. Given 1,000,000 USP units vitamin A.

April 3: Given 1,000,000 USP units vitamin A.

Laboratory data: See Table VII.

C-10. Jersey Cow.—First seen April 3, 1944. She was not eating. Was corralled in a dry lot with little or no pasture. No specific symptoms. Calf about three weeks of age. Given 1,000,000 units vitamin A.

Laboratory data: See Table VII.

C-11. Jersey Cow.—First seen April 3, 1944. Eight years old. Fed cottonseed meal, hulls, and grass hay. Last 30 days hay and bran fed exclusively. Calf one week of age.

Symptoms: Those of metritis.

Treatment: 1,000,000 USP units vitamin A and 25mg diethylstilbestrol.

April 4: Cow normal (owner's report).

Laboratory data: See Table VII.

C-12, C-13, C-14, C-15. Jersey Cows.—Corral fed animals with no pasture. All in the same herd. Fed cottonseed meal, yellow corn and alfalfa hay. Moderately good feeding practice, probably variable. C-12 was very old, C-13 was a small heifer with a calf about two weeks of age. She had experienced dystocia and was emaciated. C-14 and C-15 were apparently normal, average dairy cows. For laboratory data see Table V.

C-16. Jersey Cow.—First seen April 6, 1944. Diagnosis: Plant poisoning.

C-17. Apparently Normal Cow.—First seen April 6, 1944. Some pasture. Same herd and same feed as No. C-6.

C-18. Apparently Normal Cow.—First seen April 7. She was on fair pasture. Calf about one week of age. This farmer has had trouble in his herd from acetonemia, but there were no cases at the time of our visit. His cows were fed cottonseed meal and hulls, commercial dairy feed and peanut vine roughage. She was given 1,000,000 USP units vitamin A.

Laboratory data: See Table VII.

TABLE VII—MISCELLANEOUS DATA

	Blood				
Cow No.	Sugar Mg%	Acet. Mg%	Carot. I.U.	Vit. A per 100cc	Urine Acet. Mg%
C-4	48.5	tr	1500	12	225.0
C-5	59.0	18.2	....	..	141.0
C-7	66.0	...	1852	29	69.0
C-8	...	...	....	..	13.0
C-9	...	...	....	..	244.0
C-10	...	...	....	..	22.0
C-11	...	...	....	..	209.0
C-18	48.5	tr	....	..	....
C-19	53.0	tr	....	..	....

C-19. Apparently Normal Jersey Cow.—First seen April 7, 1944. Feeding, condition, etc., were same as for C-18. Calf about three weeks of age and scouring badly. Given 1,000,000 USP units vitamin A. The calf also was given 50,000 units of vitamin A and made a prompt recovery.

Laboratory data: See Table VII.

Discussion

The 19 cows described in the foregoing may be considered in four groups. One group, comprising cows 2-C and 3-C were primary, uncomplicated cases of acetonemia.

The second group, C-1, C-6, and C-11, were cases of metritis complicated by acetonemia.

The third group were clinically normal.

The fourth group is a miscellaneous one.

Primary Acetonemia Group

The cows in the first group had low blood sugar and high ketones in the blood and urine. The clinical laboratory data agree reasonably well with values previously reported in the literature describing acetonemia and ketosis.

These cows received no treatment other than vitamin A. The blood sugar was increased 100% in C-2 and 54% in C-3, within a period of one week. The acetone bodies in the blood of C-2 decreased from 64.6 to 2mg% in five days, and in C-3 from 73 to 2.5mg% in seven days. In C-2 the clinical symptoms disappeared completely in 24 hours and in C-3 in three days.

This is a greater average increase in blood sugar and a greater average reduction in blood ketones in a shorter period from less treatment than has been reported by other investigators.

Furthermore, the heightened blood sugar

level was not due to the introduction of dextrose from outside the body of the animal.

Metritis Complicated by Acetonemia

The animals in the second group, C-1, C-6 and C-11, were not well suited for the study of vitamin A deficiency. The metritis was probably the primary ailment in all cases. It is likely the animals were on the margin of vitamin A deficiency and when metrities ensued the acteonemia followed as a secondary

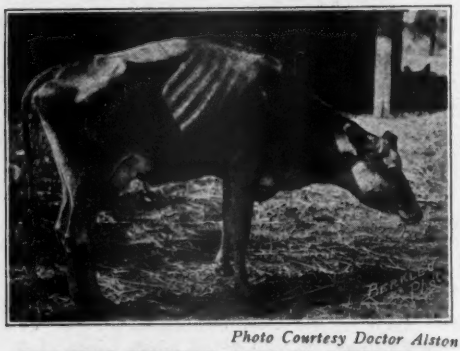


Photo Courtesy Doctor Alston

THE FIRST REPORTED CASE OF ACETONEMIA
J. T. Alston, Tupelo, Miss., was first, in the United States, to describe acetonemia in a report of the case shown above (*Vet. Med.*, 28:4, p. 159, 1933). The doctor treats about 1000 cases a year and regards the disease as one of inadequate nutrition. A varied diet of nutritious feeds he finds to be a successful preventive

matter. The cases, however, possess clinical interest. The clinical symptoms of acetonemia were relieved promptly by the administration of vitamin A but the blood picture did not clear up until the primary ailment was brought under control. It seemed obvious that vitamin A, by ameliorating the acetonemia, contributed materially to the success of the treatment for metritis and to the promptness with which recovery occurred. This was true also of similar cases, of which no examination of the blood was made and no record kept, since they were not regarded as suitable cases for the study of primary vitamin A deficiency. In the cases included in this group the study was started before the metritis was discovered.

It is to be expected, in a country and at a time when cows are low in vitamin A, that cases of intercurrent disease would be benefited by vitamin A. In the case of C-1, the blood and urine acetone level was considerably decreased by administration of vitamin A. The cow had received one ounce of chloral six hours prior to the first administration of vitamin A, but it is not likely that this would account for the reduction of acetone in the blood and urine up to the point where she was given diethyldibestrol. It is to be observed further that the clinical symptoms, with exceptions of slight inappetence and slight vaginal discharge, disappeared completely in 48 hours. She made an uneventful recovery in 10 days, and was completely normal thereafter according to later report by the owner.

It is difficult to interpret the blood chemistry changes in C-6. She responded to the vitamin A and diethylstilbestrol treatment, both in clinical symptoms and in the blood picture, for four days and then relapsed so far as blood chemistry was concerned, but the clinical symptoms of acetonemia remained in abeyance. Whether the onset of estrus was merely coincidental or whether it bore a causative relation to the apparent relapse is speculative.

It may be noted in this, and it was observable in other cases, that the Ross urine test was not a dependable indicator of the urine chemistry.

C-11 responded essentially as C-1. This cow was 25 miles distant from the laboratory, so a close check on her was not practicable. She was clinically normal in 48 hours and the blood picture was improving. A report from the owner three days later was that she was perfectly normal with milk production good.

Clinically Normal Group

It seemed desirable to ascertain the normal blood picture in cows in the same locality and environment as the affected animals. For this

Even mild cases of acetonemia walk clumsily

reason (C-12, C-13, C-14 and C-15) a reasonably well-fed group in a dry lot was selected. It will be noted (Table V) that their blood sugar was within the normal range but rather low. Blood acetone was practically normal, hemoglobin low and the blood carotene high in relation to the vitamin A. In a period of 48 hours, a single dose of one million USP units of vitamin A, raised the blood sugar an average of more than 12mg% in three of the four cows bringing it into the range of high normal. The amount of acetone in the urine was somewhat decreased and the hemoglobin was significantly increased. The most significant effect of the administration of vitamin A,

however, is seen in changes in the blood carotene and vitamin A. Before treatment a pooled sample gave carotene 382 and the vitamin A 81 I.U. per 100cc of blood. Forty-eight hours after the vitamin A was administered the carotene was lowered and the vitamin A increased.

It is reasonable to assume that the condition portrayed by this group prevails widely in cows on dry feed in Mississippi and other southern states, and that feeds carrying more vitamin A, such as properly cured alfalfa hay or corn silage, would be of great value to them.

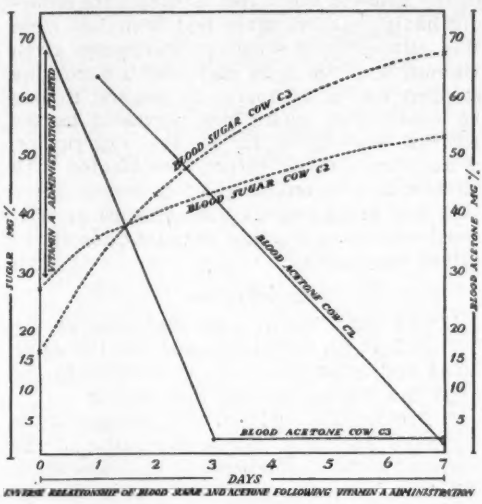
Miscellaneous Group

In C-4 the blood sugar was below normal, urinary acetone high, blood carotene very high and the vitamin A extremely low. She was not lactating and presented no clinical symptoms other than unthriftiness. The blood picture of this cow is presumably, in a measure, characteristic of the whole herd of Herefords to which she belonged—borderline cases of ketosis and vitamin A deficiency. It requires only the stress of parturition, milk production metritis or other debilitating ailment to throw such animals into acetonemia of the acute type.

Typical standing position. This animal staggered badly

C-5, the bull in this herd, on the same feed as C-4 showed a higher than the average level of blood sugar and blood and urine acetone bodies. The vitamin A content of the blood was not determined but was probably low.

C-7, of this group, was picked at random from a high-class dairy in which there was no history of acetonemia. The herd had been well fed and had increased in milk production two quarts per day per animal within the past two weeks during which time they had been on grass. The blood sugar of C-7 was in the high normal range 66mg%. The urinary acetone was slightly high. The blood carotene was very high in relation to the vitamin A. It seems significant that the blood sugar of this cow, which had been well fed all winter, was approximately the same as that of cows



INVERSE RELATIONSHIP OF BLOOD SUGAR AND ACETONE FOLLOWING VITAMIN A ADMINISTRATION

in the clinically normal group that had been on low vitamin A intake all winter. The cows in group A were not in heavy lactation as was C-7 and, therefore, their vitamin A was not depleted to the same extent as was that of C-7. It is thought the high carotene content of the blood of C-7 was attributable to the fact she had been on grass for two weeks.

Cows C-8, C-9 and C-10 also were approximately symptomless. They were dry lot cows fed cottonseed meal and hulls and poor grade hay. C-8 and C-9 were not in production; C-10 was lactating, but not eating at the time of our visit. It was not possible to perform laboratory tests other than urine examination. The acetone bodies were 13, 244, and 22mg% respectively; no albumin. They were given one million units each of vitamin A on two consecutive days. Five days later all were noticeably improved in appearance; they were brighter and the hair coat was much smoother.

The owner of cows C-18 and C-19 had had trouble with his cows in the past when they were on poor feed. Recently he had been feeding some commercial dairy feed and thought they were doing better. When seen the herd had been on pasture for two weeks. These two cows were selected at random for study. The findings were blood sugar 48.5 and 53mg% respectively; blood acetone a trace, hemoglobin 11.3 and 10.9gm per 100cc respectively.

The blood sugar and hemoglobin was low normal. One million units of vitamin A was given to each cow and the owner requested to measure the milk produced by them. He reported that they increased in milk flow and

looked better. While he had had no acetonemia since feeding commercial feed it is apparent that not enough vitamin A was being secreted in the milk for the calves as they were scouring badly (earlier calves had been lost from this ailment). The calves, also, were given vitamin A, 50,000 units each and the scouring stopped within 24 hours. In passing it may be noted that scours was prevalent in the locality and that, during the experiment, more than a dozen calves were treated with vitamin A with uniformly good results. Since this was in agreement with Phillips of Wisconsin no blood or other examinations of the calves were made.

Conclusions

1. The condition in cows diagnosed in the Cotton Belt as acetonemia present the same blood and urine picture as is reported in the literature for acetonemia and ketosis.

2. Acetonemia (ketosis with symptoms) is far more prevalent among the cattle of the southern states than elsewhere in this country.

3. The incidence of acetonemia is greatest in herds that are poorest fed in relation to their milk production.

4. Below normal blood sugar and above normal blood acetone is common in southern cattle during the dry feeding period, and many cows not in production or producing only a meager quantity of milk are on the borderline of acetonemia during the winter months, but show no clinical symptoms of the ailment, unless some depleting influence supervenes, such as parturition.

5. Low vitamin A blood levels and high carotene blood levels are common in southern cattle in the early spring.

6. Vitamin A therapy raises the blood sugar level and decreases the blood acetone level to normal in cows suffering from acetonemia and similarly affects the levels in borderline cases. It further appears to equalize the carotene-vitamin A balance in the blood; probably by improving liver metabolism which, apparently, is impaired in acetonemia and borderline vitamin A deficiency.

7. Vitamin A appears to improve the condition of the cattle (clinically and in blood chemistry) suffering from intercurrent ailments.

8. Vitamin A therapy, not only promptly dissipates the clinical symptoms and corrects the altered blood picture of animals suffering from acetonemia, but also brings the animal back to normal milk production rapidly.

9. Vitamin A increases the milk production of cows on the borderline of vitamin A deficiency and appears to increase the milk flow in clinically normal cows.

10. Vitamin A effects the foregoing changes with greater celerity than any other treatment described in the literature.

The dose recommended is one million units per cow per day and 25,000 units for calves. While a remarkable improvement is usually shown from a single dose, it is recommended that the treatment be continued for three days or longer. Excellent results were received in our earlier experiment from smaller doses, but the treatment is not expensive and, theoretically at least, there is an advantage in giving a sufficient quantity, not only to supply the immediate needs of the animal, but to enable it to store a considerable quantity of vitamin A in the liver.

Controls of course are always desirable, but this investigation was conducted in connection with a private practice and, after the results of the "new treatment" became known among the cattle owners following the use of vitamin A in the same area in February, none could be induced to forego it.

Acknowledgement.—The cooperation of Dr. W. L. Stroup, Corinth, Miss., who placed his facilities at our disposal and was untiring in his assistance, was invaluable in this investigation. We are deeply appreciative.

REFERENCES

1. Patton, John W., Acetonemia a vitamin A deficiency. *Vet. Med.*, 39:4, pp. 150-153; 1944.

2. Alston, J. T., Palsy after calving. *Vet. Med.*, 28:4, pp. 159-160; 1933.

3. Boddie, G. F., Some aspects of ketosis in dairy cows. *Vet. Rec.*, 15, pp. 1539-1546; 1935.

4. Brouwer, E., and Dijkstra, N. D., One alimentary acetonuria and ketonuria in a dairy herd induced by feeding grass silage of the butyric acid type. *J. Agr. Sci.*, 28, pp. 685-700; 1938.

5. Deuel, H. J., Jr., Butts, J. S., Hallman, L. F., and Cutler, C. H., Studies on ketosis. VII Quantitative studies on B-oxidation. Glycogen formation from various fatty acids. *J. Biol. Chem.*, 112, pp. 15-23; 1935.

6. Duncan, C. W., Huffman, G. F., and Tobin, H. A., A chemical study of ketosis in a dairy herd. *J.A.V.M.A.*, 95, pp. 690-700; 1939.

7. Fincher, M. G., Clinical observations in acetonemia of cattle. *Cor. Vet.*, 29, pp. 143-150; 1939.

8. Hayden, C. E., Fincher, M. G., and Sampson, J., Acetonemia and acetonemia with parturient paresis. *Cor. Vet.*, 29, pp. 368-376; 1939.

9. Knodt, C. B., Shaw, J. G., and White, G. C., Studies on ketosis in cattle. I. Effect of stall and pasture feeding upon the concentration of blood and urinary acetone bodies of dairy cattle. In press.

10. Lormore, R. E., Acetonemia in dairy cows. *Vet. Med.*, 29, pp. 474-476; 1934.

11. Roepke, M. H., Physiologic and metabolic aspects of acetonemia in cattle. *J.A.M.A.*, 782, pp. 411-415; 1942.

12. Sampson, J., and Boley, L. E., The physiologic basis of therapy for ketosis in the ruminant. *A. J. Vet. Res.*, 2, pp. 327-332; 1940.

13. Udall, D. H., The Practice of Veterinary Medicine, 3rd ed., pp. 312-319; 1939.

Gangrenous Mastitis in Dairy Cows*

By O. W. Schalm**
Berkeley, California

GANGRENOUS mastitis is an acute infection which appears sporadically in certain dairy herds. It develops primarily in the lactating udder but its occurrence in dry cows has been observed. As a rule it makes its appearance with remarkable suddenness. One or more quarters of the udder become swollen and very tense and, upon milking, only a few cubic centimeters of a straw-colored fluid are obtainable. The secretion from the nonaffected quarters is usually normal in appearance but greatly diminished in volume. The body temperature ranges from 103 to 106° F. There is complete loss of appetite, moderate constipation and a definite stiffness of the rear legs. In 24 to 48 hours, a bluish discoloration appears in the skin of the affected quarters. The teats are cold and insensitive to pain and, upon milking, a bloody exudate, mixed with masses of coagulated and necrotic tissue, is obtained. In some cases, gas forms in the quarter. Frequently a variable degree of edema is present anterior to the udder. The animal may die in three or four days from acute toxemia or, if it survives, a long period of convalescence follows, during which time the gangrenous tissues slough away and healing results. The appetite often remains impaired and there is a marked loss of flesh.

There are many herds in which gangrenous mastitis has never made its appearance, while in others it has become a serious problem. Parshall,[1] of California, reported that four cases developed in a dairy of 125 cows within a three-month period. In another herd of about 600 animals, 11 cases occurred within a year. Parshall made a bacteriological study of the disease and came to the conclusion that a mixed infection of *Staphylococcus aureus* and certain anaerobes, such as *Clostridium welchii*, was necessary to reproduce it experimentally. Minett[2] reported on nine cases in which he considered staphylococci to be the primary infection since, in every instance they were found while a mixed infection with anaerobes could be proved in only one. Little and Foley[3] described cases of acute mastitis due to staphylococci, one of which developed into the gangrenous form. Bean, Miller and Heishman,[4] in discussing 15 cases of mastitis which they considered due to *Corynebacterium pyogenes*, stated that, with two cows, the skin around the base of the teat showed a pronounced bluish discoloration and the rear quarters of one of the udders sloughed off completely.

The writer has made some observations and preliminary studies on gangrenous mastitis. The results obtained thus far are as follows:

Case One

This herd consisted of about 100 milking cows which had been subjected recently to a change in management. The herd was moved to a new location and the owner, who had been milking the cows, now employed others to do the work. Milking machines were used and the cows were stripped by hand. The man following the machines was very careless about getting milk on his hands and did an indifferent job of stripping. A case of gangrenous mastitis occurred shortly after the change in management; this was followed by five more cases within a period of six weeks.

The writer visited the herd two weeks after the last case of gangrenous mastitis had developed. Three gangrenous udders were selected as still suitable for culture studies. Among the twelve quarters on these udders, one was not involved, seven were swollen and contained pus and four were gangrenous, but only one of the latter was suitable for sampling. The exudates were iced while enroute to the laboratory, where veal infusion agar plates containing horse blood were prepared from each sample as well as anaerobic cultures in cooked beef heart medium.

The gangrenous exudate, and four of the purulent samples, gave rise to a very high count of minute hemolytic colonies which proved, on culture and animal inoculation tests, to be *C. pyogenes*. Among the three remaining purulent exudates, one gave rise to no growth in blood agar, another contained 380,000 staphylococci per cubic centimeter and the third gave rise to a very high count of *Streptococcus agalactiae*. No anaerobes were found in the beef heart medium, but stained smears of the growth revealed many staphylococci in every sample; two samples also contained streptococci and five showed many small rods and filaments as well as staphylococci.

a. *A trial to determine the effect of Corynebacterium pyogenes on the normal dry udder.* —Since *C. pyogenes* was the predominating organism in the blood agar plates, strains were isolated for animal inoculation experiments. No lactating cows were available and a dry animal was, therefore, selected. Five cc

*Presented at the Annual Meeting of the California State Veterinary Medical Association at San Luis Obispo, January, 1944.

**Veterinary Science Division of the University of California.

of pooled 48-hour broth cultures of *C. Pyogenes*, isolated from the individual exudates, was introduced into the teat canal of the right front quarter of Cow No. 279 and a similar quantity of pooled broth cultures of *C. pyogenes* (obtained from the organs of a mouse, previously inoculated with a culture isolated from the gangrenous exudate) was introduced into the right rear quarter. Within four hours, both inoculated quarters were markedly swollen and firm throughout, while the non-injected ones remained normal and pliable. By the second day, the swellings were receding and at that time each injected quarter contained about 50cc of a glutinous, non-odorous pus. Five days after the exposure, the udder had practically returned to normal.

Minett[2] described a case of acute mastitis in which staphylococci were the only pathogens isolated at the onset of the disease but, a month later, *C. pyogenes* was the predominating organism in the exudate. His finding suggests that, at times, this organism may serve as a secondary invader in acute mastitis and that in such instances, if culture studies are delayed, *C. pyogenes* may mask the organism of primary significance.

Case Two

A herd of about 115 milking cows in which three cases of gangrenous mastitis occurred

Fig. 1. Cow No. 306, 72 hours after the injection of pooled gangrenous exudate from field cases into the left quarters. Note edema anterior to udder

simultaneously. A year previously, a single cow had been affected with the disease. The herd was machine-milked and stripped by hand by the owner and his son. During the day, the cows were on irrigated pasture, while at night they were kept in a large corral. Recently, while irrigating an adjoining pasture, water overflowed into the corral leaving a fairly large mudhole. The owner stated that he had not observed cows lying down in the mud.

Fig. 2. Cow No. 306, rear view, 72 hours after inoculation. Note swelling and discoloration of left quarters

The three cases of gangrenous mastitis were seen within 72 hours after appearance of the initial symptoms. The left rear quarter was the primary focus of the gangrenous process in each cow. The reason for this was not apparent. Samples were collected from all quarters and were iced while enroute to the laboratory. Veal infusion agar plates containing horse blood were inoculated with material from each sample as were also beef heart anaerobic culture media. No anaerobes were found but the exudates from the three gangrenous quarters as well as the secretions from seven other quarters contained staphylococci. Typical staphylococcus colonies were transferred from the agar plates to serum broth and, after incubation, they were plated in agar containing ox blood to test for toxin production.[2] All cultures from the gangrenous quarters as well as those isolated from five other quarters proved to be staphylococci which produced both *alpha* and *beta* toxins. Further studies proved the cultures to be typical of *S. aureus*.

b. *An attempt to induce gangrenous mastitis with exudates from natural cases of the disease.*—The exudates from the three gangrenous quarters were mixed and 5.0cc was injected through the teat canal into each left quarter of lactating Cow No. 306. The following day these quarters were markedly swollen and considerable edema of the abdomen, di-

rectly anterior to the udder (Fig. 1), was present. The animal was listless, stiff in the rear legs and its temperature was 103.6° F. The udder was not painful on palpation, but the edematous abdominal wall was very sensitive. Forty-eight hours after inoculation, the left quarters were blue (Fig. 2) and contained a bloody exudate and gas. On the fourth day, a serosanguinous fluid was oozing from the gangrenous tissues. This was followed by putrefaction with complete sloughing of the left half of the udder 17 days after the exposure (Fig. 3). The gangrenous exudate from each quarter contained a very high count of both *S. aureus* and *Str. uberis*, but was devoid of anaerobes. The animal was slaughtered on the 25th day because of advanced emaciation.

c. *First trial to produce gangrenous mastitis with whole-broth cultures of Staphylococcus aureus.*—Ten strains of toxin-producing staph-

Fig. 3. Cow No. 306, 17 days after inoculation. Left half of udder has dropped off, leaving stumps of large blood vessels protruding from abdominal wall

ylococci, isolated from the gangrenous quarters of the three field cases, were grown in broth for 24 hours at 37° C. and then mixed. Five cc of the pooled cultures was injected into the left front quarter of lactating Cow No. 1 and 2.5cc was introduced into its left rear quarter. Twenty hours later, both quarters were markedly swollen and were turning blue. The hind legs were stiff and the animal was reluctant to walk. Anorexia and constipation were evident and the temperature was 104° F. The affected teats contained the typical bloody exudate without gas. The following day an extensive edema of the abdomen was present. The gangrenous exudates, 24 hours after inoculation, contained pure cultures of *S. aureus*. The two left quarters sloughed away on the 24th day, after which the animal made an uneventful recovery.

d. *First trial to produce gangrenous mastitis with washed broth cultures of Staphylococcus aureus.*—The rapidity of development of the gangrenous process in Cow No. 1 suggested the probability that toxins in the broth cultures were responsible for immediate damage to the mammary tissues which led to the early appearance of gangrene. To determine whether this might be the case, the ten cultures of *S. aureus* were grown in broth for 24 hours at 37° C. They were pooled, centrifuged, and the broth replaced with sterile physiological saline. Since no lactating cows were available, dry Cow No. 285 was used and it was given 2.5cc of the pooled, washed cultures in each left quarter. The following morning the animal was eating and was active, although there was a moderate swelling of the injected quarters. The udder contents were watery and there was no evidence of gangrene. On the third day, the swellings had decreased, but the inoculated quarters now contained a purulent exudate having a comparatively low count of *S. aureus*. The cow was turned out to pasture and was not examined again until three months later. At that time, the animal was still dry and no difference could be detected between the right and left halves of her udder. Each quarter contained a serous secretion and that from each left quarter showed a very high count of *S. aureus*. Although gangrenous mastitis had not been produced, a persistent infection with *S. aureus* had resulted from the exposure.

e. *Second trial to produce gangrenous mastitis with whole-broth cultures of Staphylococcus aureus.*—Since the washed cultures of *S. aureus*, resuspended in saline, had failed to induce gangrenous mastitis in a dry cow, another dry animal, Cow No. 288, was exposed to whole-broth cultures. Pooled 24-hour cultures were used and 8cc was introduced into the right front quarter and 5cc into each of the left quarters. The following day these quarters were moderately swollen and contained a thick yellow pus in which *S. aureus* was present in pure culture. The temperature was 103.4° F., but the animal was active and eating. After the third day, the swellings had begun to recede and the cow was turned out to pasture. Two months later, it calved normally; the udder showed no visible symptoms of mastitis, but *S. aureus* was present in the milk from the three previously exposed quarters.

f. *Third trial to produce gangrenous mastitis with whole-broth cultures of Staphylococcus aureus.*—Failure to produce gangrenous mastitis in the two dry udders with either washed or whole-broth cultures of *S. aureus* suggested that probably the parenchyma of the dry udder is less susceptible to the destructive action of the staphylococci than that of the

lactating udder. Therefore, two normal lactating cows were purchased for further trials. One of them, Cow No. 758, received 5cc of pooled 24-hour broth cultures into each right rear and left front quarter while the right front quarter was given 5cc of a 48-hour growth. Three hours later, the animal was shivering, its temperature was 103.4° F. and the injected quarters were markedly swollen and firm. The following morning the animal was down and showed reluctance to get up. Two quarters were turning blue and contained the typical bloody exudate. On the second day, all inoculated quarters were gangrenous and the process was spreading to involve the one noninjected quarter also. By the fourth day, the entire udder was swollen and gangrenous and an extensive edema of the abdomen anterior to the udder had developed. During the next four weeks the udder underwent putrefaction (Fig. 4) and finally sloughed away, leaving an extensive denuded area. The animal was emaciated and since there was little hope for its recovery, it was destroyed.

g. *A trial to determine the effect of toxins contained in bacteria-free filtrates of broth cultures of Staphylococcus aureus on the udder.*—The 10 strains of *S. aureus* were grown in broth at 37° C. for 72 hours and pooled. One-half the volume was filtered through a No. 5 Mandler filter and the other half through a Berkefeld V filter. Lactating Cow No. 732 received 15cc of the Mandler filtrate into its right front quarter and 20cc of the Berkefeld filtrate into its right rear quarter. Four hours later, both quarters were distinctly swollen (Fig. 5) and the body temperature was 103.2° F. The following morning the swellings were decreasing and at that time the injected quarters contained a sterile yellow secretion which formed a heavy sediment on standing. By the third day, the udder and its secretion had returned to normal.

Case Three

A herd consisting of approximately 110 cows which were machine-milked and stripped by hand afterwards. The owner was seeking help because some of the animals intermittently developed one or more swollen quarters and, recently, a case of gangrenous mastitis had occurred in one of the cows. Arrangements were made to collect composite samples from all lactating animals.

A search was made for streptococci and staphylococci, using the Hotis test, microscopic examination of incubated milk, isolation of colonies from modified Edwards' medium[5] streaked with incubated milk, and plating the milk, before incubation, in veal infusion agar containing ox blood. A rather unusual result was obtained for *Str. agalactiae* was not found in a single sample, but two cows were shedding *Str. uberis* and 10 samples contained atypical streptococci. Twenty-one of 89 cows tested were found shedding toxin-producing strains of *S. aureus*.

h. *A trial to determine the effect of Staphylococcus aureus, isolated from nongangrenous udders, on a normal lactating udder.*—Eighteen strains of *S. aureus*, isolated from the milk from nongangrenous udders, were grown in broth for 24 hours and pooled. Lactating Cow No. 790 received 2.5cc of the pooled broth cultures in her right front quarter and 5cc in

Fig. 4. Cow No. 758, 20 days after inoculation of three quarters of the udder with broth cultures of *Staphylococcus aureus*. All quarters are gangrenous and are undergoing putrefaction and sloughing

her right rear quarter. Four hours later, both quarters were swollen and very firm. The following day the cow was eating although both right quarters were markedly swollen and her temperature was 105.4° F. On the second day, the teat and ventral surface of the right front quarter were gangrenous and only a small amount of straw-colored fluid could be obtained. The gangrenous process remained localized, and five weeks after the exposure, the teat and tissues around its base sloughed away. The right rear quarter was swollen and contained a thick pus but showed no signs of gangrene. Later, a fistulous tract developed through which the purulent exudate drained. While a violent case of gangrene had not occurred in this trial, a localized gangrenous process had been produced with cultures of *S. aureus* isolated from nongangrenous udders.

i. *A trial to determine the effect of exposure of the udder to staphylococcus toxins in the form of bacteria-free broth filtrates upon a subsequent inoculation with virulent Staphylococcus aureus.*—Cow No. 732, which, six weeks previously in trial "g", had developed a transitory mastitis following the injection of bacteria-free filtrates of broth cultures of *S. aureus* into two of its quarters, was injected

with the same pooled cultures used to infect Cow No. 790 in experiment "h." Five cc of the pooled cultures was injected into each of the right rear and left front quarters. The following day both quarters were swollen and contained a purulent exudate. The animal was active and eating; her temperature was 101.3° F. The swellings were found to be receding on the second day and by the third day there was little difference in size between the inoculated and control quarters. This cow was approaching the end of her lactation period and was not milked after this—a month later the udder was examined again. The inoculated and control quarters showed no obvious differences and all contained a serous secretion. Agar plates containing ox blood revealed 20,000 toxin-producing staphylococci per cc in the right rear quarter while the left front quarter contained less than 100 colonies per cc. The results of this trial suggest that the previous exposure of the udder to bacteria-free filtrates of broth cultures of *S. aureus* had increased its resistance to that pathogen but did not prevent the organism from becoming established within the udder.

Discussion

Gangrenous mastitis is a serious disease because the affected cow either dies in a few days or, if it survives, the udder is usually so severely damaged that the animal loses its value for dairy purposes. Studies on the etiology of this form of mastitis have been meager but the few reports that have appeared point to *Staphylococcus aureus,* or a mixed infection with this organism, as the primary cause.

Changes in the udder which lead to gangrene apparently begin within a few hours after the first symptoms appear. It is then too late for therapy to save the affected quarters. The disease must be controlled through preventive measures, and this will be possible only when the etiology and other factors involved are thoroughly understood.

Corynebacterium pyogenes was found to be the predominating organism in the exudates from three cases of gangrenous mastitis in one herd. Bacteriological studies were not made until two weeks or longer after the first symptoms had appeared in these cows; thus it is possible that *C. pyogenes* was a secondary invader and masked the organism of primary importance. Pure cultures of *C. pyogenes* isolated from these cases failed to induce gangrenous mastitis when injected into a normal dry udder.

Toxicogenic strains of *S. aureus* were isolated from the gangrenous exudates obtained early in the disease from three cases of gangrenous mastitis occurring simultaneously in another herd. A violent form of gangrenous mastitis was produced in two lactating cows following injection of 2.5 to 5.0cc of pure broth cultures of these strains of *S. aureus* into the teat canal. Two dry udders receiving similar injections developed only a transitory mastitis. These results suggest that the lactating udder is more sensitive to the destructive action of the staphylococci than the dry

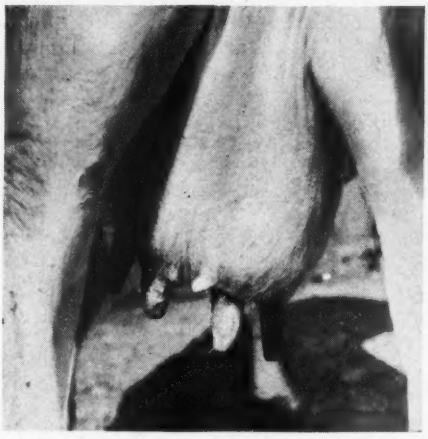


Fig. 5. Cow No. 732, rear view, after injecting bacteria-free broth filtrates of cultures of *Staphylococcus aureus* into the right quarters. Note swelling of right half

udder. The gangrenous process probably develops as a result of injury to the vascular system of the udder followed by thrombosis of the vessels. The lactating udder is more vascular than the dry udder and it is possible that this difference renders it more vulnerable to the development of the gangrenous form of mastitis.

The rapidity with which gangrene appeared following the injection of broth cultures of *S. aureus* into lactating quarters suggested that toxins, elaborated by the organisms during incubation of the cultures, were responsible for immediate injury to the vascular system. However, toxin alone in the form of bacteria-free filtrates of broth cultures of *S. aureus* failed to produce gangrenous mastitis in a lactating udder. This exposure to toxins in the bacteria-free broth filtrates, however, appeared to impart some resistance in the udder to a subsequent exposure to virulent *S. aureus.*

Toxicogenic strains of *S. aureus,* isolated from milk of normal appearance, produced by cows in a herd in which a case of gangrenous mastitis had occurred, induced a localized gangrenous process in one quarter and an acute purulent mastitis in another quarter when broth cultures were injected into the teat canal. This result suggests that cows may

harbor pathogenic strains of staphylococci in their udders without the occurrance of gangrenous mastitis but that when resistance of the udder becomes lowered, the staphylococci may grow rapidly and produce gangrenous mastitis.

Conclusions

It is apparent that the etiology and the methods for the prevention and the treatment of gangrenous mastitis need further study. *S. aureus* has been given a place of primary importance, but it is not certain that other pathogens cannot serve as etiological agents. It is necessary, therefore, to make bacteriological study of many cases of the disease in order to establish more definitely the primary cause. Since *S. aureus* appears to be of importance, a thorough investigation of the entire staphylococcus problem as it pertains to mastitis is indicated.

REFERENCES

1. Parshall, C. J., Natural and experimentally produced gangrenous mastitis in cows. *Cor. Vet.*, 24, pp. 146-155, 1934.

2. Minett, F. C., Studies on bovine mastitis—XII—mastitis due to staphylococci. *J. Comp. Path. & Therap.*, 50, pp. 101-121, 1937.

3. Little, R. B., and Foley, E. J., Staphylococci associated with mastitis. *J.A.V.M.A.*, 87, pp. 637-649, 1935.

4. Bean, C. W., Miller, W. T., and Heishman, J. O., A note on *Corynebacterium pyogenes* as the cause of bovine mastitis. *J.A.V.M.A.*, 103, pp. 200-202, 1943.

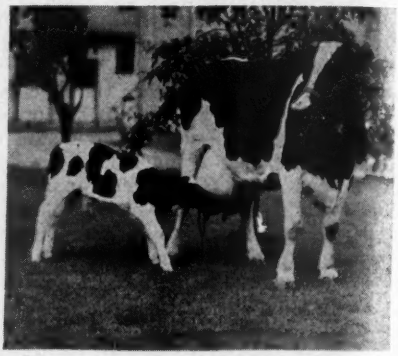
5. Plastridge, W. N., Anderson, E. O., and Weirether, F. J., The control of *Streptococcus agalactiae* mastitis by a segregation program based on periodic laboratory tests. Sup. to Storrs Agri. Exp. Sta. Bul. 240, 1942.

Breeding and Fattening Cattle Need Vitamin A

Vitamin A deficiency is a common cause of reproductive failure in beef cows, birth of weak calves that fail to survive, impotency in bulls, and incoordination and generalized edema (anasarca), or swelling of the legs, brisket, shoulders and other parts of the body of breeding and fattening animals.

In vitamin A studies with beef cattle to determine the requirements for reproduction, 12 calves were born to cows maintained on only 1,360 to 2,275 micrograms of carotene per 100 pounds of body weight. Of these 12 calves, four were dead at birth, three were weak and blind, one was weak but not blind, two were rated as fair and two good at birth. In contrast to these results, of 16 calves born to cows receiving 2,725 to 10,900 micrograms of carotene per 100 pounds of body weight, eight were rated as excellent, three very good, three good, one weak, and one died at birth.

A study showed that vitamin A deficiency in bulls adversely influences sexual desire and activity of the animals as well as the quality of the semen. In three months, two of three bulls were so depleted of vitamin A that within the next month one was unable to give service and became blind. The other gave service for a longer period, but all the spermatozoa soon became abnormal and no motility was observed. After missing a few services, this bull was fed vitamin A with the result that he was soon able to give service regularly. The percentage of abnormal spermatozoa decreased, and motility improved. Apparently the injury is permanent, however, since more than 50 percent of the spermatozoa continue to be abnormal and motility is low. No response was noted from the administration of vitamin C to vitamin-A-deficient bulls, but definite improvement followed carotene feeding.



Johanna Hester Prilly at the age of nine years. In 1938 she broke the then world's record by producing 41,414 pounds of milk; netting her owners $2,070.70 and a calf valued at $5,000 at birth. Her butterfat yield was 1,221.4 pounds. She was milked four times daily. Her daily ration was well adapted to her superlative production; consisting of 36 pounds of concentrates (rolled oats, mill run, dried beet pulp, alfalfa meal flavored with molasses, cocoanut meal and linseed meal), 60 pounds of alfalfa hay, 5 pounds oat hay, and two hours on pasture. It will be noted that many of the ingredients of this ration are rich in vitamin A

Vitamin A deficiency was found to be prevalent in fattening cattle in feed lots where distillery slop or mash and low-quality roughages were fed. Feeding good-quality hay or fish-liver oil appeared to cure affected animals and to prevent further cases of this deficiency. Indications are that vitamin A deficiency can be prevented in beef cattle by providing breeding animals with five to 10 pounds of good-quality hay per head daily and fattening cattle with three to five pounds of hay or one-half to one pound of high-grade alfalfa leaf meal, or approximately 25,000 international units of vitamin A in oil or concentrate form.—Ann. Rept., B.A.I., U. S. Dept. Agr., 1943.

Control Bovine Brucellosis Now

By S. L. STEWART
Olathe, Kans.

A DEMAND for brucellosis control and eradication is growing throughout the cattle industry. Confidence, on the part of the cattlemen that the disease can be controlled, is growing also. Two things stand in the way.

1. Archaic regulations of some livestock sanitary officials governing the importation of cattle in their respective states.

But these regulations are not always as dumb as they look. Some of them are not designed primarily for the control of disease among animals but mainly to give "home grown" cattle breeders an advantage over those living in other states. Disease control features of such regulations are merely for the purpose of bringing them within the police power of the state.

Fortunately some livestock sanitary commissions have gone beyond the Bureau's regulations and made their own rules. In Massachusetts, for example, selection of the animals to be vaccinated, young or adult, is left to the decision of the veterinarian who has the herd in his care and who knows more about what is needful for it than any one else.

2. Unrealistic federal attitude and regulations for payment of indemnities.

The B.A.I. is on a spot in making these regulations. Having undertaken the eradication of bovine brucellosis without sufficient information about the disease, it adopted an unworkable plan. Unfortunately, it went out on a limb in the matter of opposing vaccination for the prevention of the disease. When the time arrived that the Bureau was forced to adopt vaccination as a part of the plan of brucellosis control or see congressional appropriations shut off, of course, it was adopted; thus, in effect, admitting that it had mistakenly forced the unnecessary slaughter of hundreds of thousands of valuable dairy and breeding animals. How we need them and their progeny now! To dull the edge of admission, a new word "calfhood" was coined to describe the vaccination, which must have succeeded beyond the fondest hopes of the authors. Apparently many assumed it must mean something different from "calf" although, obviously, when one came to a farm to do the vaccinating only a calf could be found—search as they might. So generally the calf was vaccinated.

This limited acceptance of vaccination constituted a big improvement in the plan of control, mainly because a large number of veterinarians, perhaps because they were unable to find "calfhood" in their dictionaries, interpreted it to mean any bovine animal not beyond the age of usefulness for breeding or profitable milk production. The wisdom of this interpretation was apparent immediately in the improved results attained and was proved conclusively by research veterinarians at the California experiment station. They demonstrated that vaccination before the animal is sexually mature is not nearly so effective as vaccination afterwards. The minimum age at which heifers of the dairy breeds should be vaccinated was set, by the California investigators, at eight months and for beef breeds one year. Dropping the emergency word "calfhood" and merely saying vaccination is overdue.

The high-grade Holstein cow, shown above with her owner, was 15 years of age when the photograph was taken. She had produced 12 normal healthy calves and approximately 160,000 pounds of milk and 6,000 of butterfat. She calved the first time at three years of age and retained her afterbirth. She was given a blood test for brucellosis and gave a positive reaction in three dilutions. I vaccinated her with live culture immediately and also when it was certain that she was again pregnant. She reacted, positively, for the next 12 years and was positive in dilutions of 1:100 at the time the photograph was taken. In all this time she has been a member of a negative herd in which no other reactors have developed. Comment is unnecessary

The price of dairy cattle is extremely high

and all the milk that can be produced is urgently needed by the war effort. To slaughter a high-producing cow that is a regular breeder and non-infective, because she has, at some time, been infected by Brucella organisms and still gives a positive reaction to the agglutination test, is an act of sabotage. Beef cattle of high breeding value are equally needed to procreate our future food supply and they are meeting the same fate.

Everyone knows that in herds, where a small number of animals are infected and the infection is quiescent, the disease can be eradicated by blood testing and elimination of the reacting animals. But slaughtering profitable producers that react is something else again. That is why I say that regulations are archaic and unrealistic.

Every veterinarian knows that in herds where the infection is wide-spread and virulent—a brucellosis "storm" occurring—the test and slaughter plan will prove costly in the extreme and is likely to destroy the herd and bankrupt the owner. And every veterinarian, who has not closed his mind to facts that controvert preconceived opinions, knows that the majority of such herds can be saved and rendered profitable by vaccination and indicated corollary sanitary measures, and that the disease can be eradicated without sacrificing a profitable animal. But it can't be done by vaccinating only the "calfhoods," i.e., until they replace the producing herds.

The cattlemen of the United States and Canada are anxious to get rid of brucellosis and keep it out of their herds. They are leading the way. This was amply evident at a cattlemen's meeting in Denver, which the writer attended in February where there was a universal demand for regulations under which this work can be handled.

Distomiasis of Cattle*

The loss due to the liver fluke in cattle in this country is enormous. The condemnation of livers due to these parasites at the packing houses having federal inspection is 1.2% of all livers. For the whole United States this would mean one and one-half million livers. To this must be added loss of flesh, loss of milk, loss due to curtailed breeding and from mortality. The total loss is computed to be the equivalent of the loss of 10,000 animals annually.

The snail host to the liver fluke is practically omnipresent in the coastal prairies of Texas. It lives at the edge of temporary pools, where there is a great fluctuation of the water level. Cow tracks about the margin of temporary and permanent pasture ponds are a favorite habitat of the snail and the over-flow from watering troughs and flowing wells supplies a favorable site for its development. The borrow ditches along highways are artificial incubators for flukes. As many as 200 snails per square foot have been collected about the margin of ponds that were drying up. On an average, 0.5% of the snails carry the fluke infection.

The snail eggs are laid in the temporary pools where they hatch throughout the mild winters of the Gulf Coast region. The fluke eggs, which are passed in the droppings of the cattle, hatch in the same pools with the snails.

The newly hatched animal is called a miracidium; these penetrate the body of the snail where, in the course of about seven weeks, they develop cercariae. A single miracidium may produce as many as 650 cercariae. The cercariae escape from the snail and quickly encyst on vegetation or even upon the surface of the water. After encysting the cercariae are ingested by cattle, whence they escape from the intestine by penetrating the intestinal wall. They migrate to the liver and enter it through the hepatic capsule. The adult stage is developed within the liver.

Carbon tetrachloride is an effective remedy for flukes, but it is dangerous for cattle and its use is inadvisable in these animals.

Hexachloroethane is a satisfactory treatment for fascioliasis in cattle. It is best administered in the form of an aqueous suspension as a drench. The suspension may be prepared as follows: Thoroughly mix 500 grams of commercial (brown) or white hexachloroethane and 50 grams of powdered bentonite. To this mixture add slowly, with constant stirring, 750cc of water; the final mixing is accomplished with an electric kitchen mixer. This results in a suspension bulking 1000cc. It may be administered with a dose syringe or by drenching, at the rate of 200cc for each adult animal and 100cc for a calf. More than 3700 animals have been treated in this way with a loss of only six. The cattle treated included range stock of all types and kinds, young and old, fat and thin, weak and strong. The six that died were extremely debilitated, almost moribund when treated. Weak animals may go off feed for a day or two after treatment. Vigorous animals are not visibly affected. When given according to the above-mentioned doses, hexachloroethane suspension has proved highly effective in killing the common liver fluke in cattle.

*Notes from an address O. Wilford Olsen (B.A.I.), at the 13th Annual Conference for the Louisiana Veterinary Medical Association by the Louisiana State University and A. & M. College, Feb. 16-17, 1944.

Role of the Veterinarian in Poultry Diseases*

J. F. HARR**
Sacramento, California

THE veterinary profession is constantly being apprised of the fact that veterinarians are urgently needed to serve the poultry industry. The need is obvious when we consider that the annual poultry mortality throughout the nation is estimated by several writers to be between 30 and 40%. Poultry raising is an important industry, producing vital food products, and its needs demand serious consideration. The agricultural history of this country bears out the fact that diseases which have seriously threatened our livestock industry have been successfully controlled by the veterinary profession.

Various programs have been suggested for the eradication and control of poultry diseases. These have received wide publicity in veterinary journals and livestock publications.

The veterinarian, as any other professional man, has the right to expect a just remuneration for his services. It is with this thought in mind that I believe it would be wise to discuss briefly the growth, size, wealth and distribution of the poultry industry in California; and also the type of service this industry has received in the past.

The poultry industry, aided greatly by rapid advances in the artificial incubation of eggs, brooding of chicks and poults, coupled with better management practices and advances in nutrition, has made a phenomenal growth.

In 1942, this state produced 19,871,000 chickens and 3,137,000 turkeys. The California poultry industry produced in 1942 a cash crop worth $90,477,000 consisting of chicken eggs $49,130,000; chickens $10,987,000; broilers $6,396,000; turkeys $18,964,000; and turkey eggs $5,000,000. On January 1, 1943, there were 13,659,000 laying hens on California farms. Figures for 1943 are not yet available, but the crop was larger and its cash return to the farmer will exceed the 1942 figure.

In studying the poultry industry of this state it is amazing to find that, of the 58 counties in the state 20 have a sufficiently sizable poultry population to support one or more veterinarians working exclusively with this branch of the livestock industry.

With the commercialization of the poultry industry the disease problem became one of its greatest limiting factors. When this was first recognized the industry was overrun with many would-be poultry experts, quacks and medicine peddlers, and millions of dollars were spent for useless remedies. As this type of disease control proved valueless, and research in poultry diseases began to reveal the fact that fowl pox, laryngotracheitis, pullorum disease, and such poultry parasites as coccidia, lice and roundworms could be successfully controlled, poultrymen began to seek better information and advice.

Veterinarians, except in a very few locations, have not taken an active interest in poultry diseases. As a result of this situation there have developed a few laymen who have become quite proficient in aiding poultrymen with some of their disease problems. Some of these men are vaccinating birds for fowl pox and laryngotracheitis, and conducting the rapid whole blood agglutination test for pullorum disease. Further these men have acquired a good general understanding of the poultry industry and its problems and have aided the poultrymen in some of their management problems. It may be that these laymen will figure prominently in future activities pertaining to poultry disease. They are now, in some instances, assisting the veterinarians much in the same manner as the nurse technician aids the physician. It would appear that the veterinarian, with his training, can serve the industry in a manner that cannot be equalled by any other professional or lay group. This is true in spite of the fact that some laymen have demonstrated ability in vaccination and the rapid whole blood testing for pullorum disease.

Services that the veterinarian can give the poultryman, and the poultry industry's greatest needs are: A prompt accurate diagnosis, sound practical advice and recommendations relative to poultry hygiene, sanitation, management and nutrition. These needs are many and exacting and require a lot of thought, time, and study on the part of the veterinar-

*Presented before California Veterinary Conference San Luis Obispo, January 5, 1944.
**Division of Animal Industry, California State Department of Agriculture.

ian. He must not only become familiar with the various poultry diseases, but must possess a wide knowledge of the poultry industry.

That the veterinarian can serve the poultry industry in this way to the benefit of both has been demonstrated by the fact that, throughout the country and in this state, veterinarians, as research and public service veterinarians, practitioners and others, are working exclusively with poultry diseases. We have recently witnessed a definite increase in the number of practitioners in this state who are devoting part or full time to a poultry practice. The activities of such men have done much for the industry and have increased the demand for such veterinary services.

Poultry diseases present a little different problem than other livestock diseases. This is due to the fact that the economic value of the individual bird is in most instances small, and does not justify individual treatment; and experience has shown that the most successful way to control poultry disease is by preventive measures.

The industry is located in various centralized districts throughout California and large numbers of birds will be found in these areas. Various marketing associations, cooperatives and other allied industries, such as feed manufacturers, egg sales agencies and hatcherymen, have brought together groups of poultrymen in these areas as business units, funtioning as a group. In many instances these organizations are giving much financial aid to the poultrymen of their group or association, and developing policies for these groups and representing them in many ways.

In recent years the leadership of such groups has become more progressive. They are interested in better disease control methods and are definitely seeking better advice and information. This development affords the veterinarian an unusual opportunity. For one thing, it offers a better opportunity for the establishment of disease control through a group—thus saving much time—as the development of such programs through the individuals is laborious, time consuming and expensive. Further, it could provide the veterinarian volume to work with, insuring a definite income for his services.

The importance and newer knowledge of poultry diseases, particularly pullorum disease, has offered opportunities for several veterinarians in this state to engage in poultry practices, centered around well planned and organized programs. These have proved beneficial to both the veterinarians and the industry.

In 1939 the California State Department of Agriculture offered to the industry a voluntary official pullorum disease program, which is now in operation in four areas in the state. There are approximately 230,000 birds entered in this program. Practicing veterinarians are used as inspectors for these areas. Committees in these areas, representing the poultrymen, have selected various veterinarians who after a written examination, have been approved by the State Department of Agriculture as official inspectors. They are paid by the poultrymen for their services and all arrangements for such services have been worked out between the various committees and the inspectors. To date all the inspectors are obtaining the same fee.

The volume of work that such a program provides gives the poultryman a dependable service and the veterinarian a secured livelihood and it, also, affords him an opportunity to serve a single industry to the advantage of both. It further provides a complete veterinary service for the industry.

The poultryman uses such veterinary service to secure prompt, accurate diagnoses and advice in the prevention and control of his poultry disease problems. He can obtain better biological products, advice as to their use and service in the administration of such products. This is equally true of the few drugs that are of value in the treatment of poultry diseases and parasites.

As has been previously mentioned, the small economical value of individual birds necessitates that the veterinarians have a large number to work with. To accomplish this much thought and planning must be given to any disease control program or project undertaken. As the result of such planning and organization the veterinarian has the knowledge as to when the greater part of the work is to be done. Therefore, he can arrange for a satisfactory crew to handle and assist him in his work.

To give a concrete example of how this works out: last fall a veterinarian doing poultry practice and conducting a large amount of blood testing was able to schedule a full day's work for himself and a crew of four people for 90 consecutive working days. Not only did it benefit him, but it also aided the farmers in planning their work and securing the necessary labor to do the job.

In many instances, ranches in a particular location were lined up for succeeding days so that these ranchers could exchange help. This further provides a crew that is accustomed to working together and has developed speed in handling birds. In many instances blood samples were obtained from three to four thousand turkeys per day.

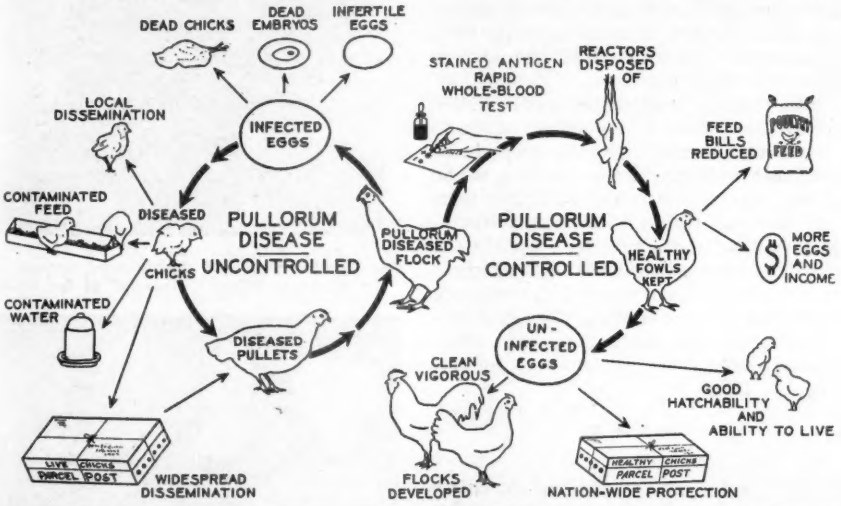
In building a poultry practice the best advertising is good service and the success attained. It is pleasing to note that in areas

where veterinarians have taken an active interest in poultry diseases, outstanding satisfaction has resulted. Some of these accomplishments are improved ranches, better ranch practices and better stock; in addition there has been a definite decrease in mortality from disease.

To illustrate what can be achieved in a well organized progam, one project in this state might be mentioned where unusually satisfying results have been obtained. In 1942 a group of turkey growers in one large producing area in this state experienced a very high mortality, averaging 30%, in poults up to four weeks of age. The next year these growers, as a group, decided to purchase only poults from those breeding flocks the owners of which were participating in the California official pullorum disease program i. e., pullorum passed flocks. To qualify for this grade they must have passed one complete test. There were 535,000 poults brooded on 89 different ranches and no losses were traceable to pullorum disease and the mortality from all causes, for the first four weeks, averaged 12%. Losses up to marketing time were greatly reduced as compared to those of the previous year. The birds were better developed and better finished and the farmer received greater returns.

From this project, in 1943, were selected approximately 45,000 breeders, which were negative on the first test for pullorum disease. With better management practices and housing of breeder birds, many of the breeders selected are now in full production.

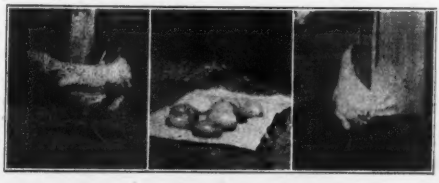
I would not want to intimate that poultry practices can be built only with the aid of organized groups of poultrymen. There is one area in this state where two veterinarians have developed very successful poultry practices by working with individual poultrymen. In this area approximately five million chickens are produced of which 90% are vaccinated by these veterinarians. They can do the job cheaper and more efficiently than the poultryman himself could do it. This has resulted from their knowledge of the poultry industry, of the way to handle the birds effectively, and of how to conduct their practices in an orderly manner. Both veterinarians use an experienced crew to help them handle the birds. In this area there are very few laymen doing vaccination and other similar services.



Comparison of injurious effects of uncontrolled pullorum disease with the benefits resulting from the use of the agglutination test and disposal of reactors. (From Misc. Pub. 349, U. S. Dept. of Agriculture.)

It is also remarkable to note that no serious outbreaks of laryngotracheitis and fowl pox have occurred in this area in recent years.

In closing I would like to leave with you the thought that probably in your own area there is a considerable poultry population and that the poultry industry needs your services. Your services to this industry can be of a definite value to the poultrymen and to yourselves. To many of you this may be a new venture and, to undertake this new type of practice will require thoughtful study and investigation on your part.



Clinical Reports

Mushroom Poisoning in Cattle

Plentiful rains in October, 1943, following a drought, caused a rank growth of mushrooms to spring up before the grass, which had been dried up, got started. Cattle ate these mushrooms greedily and many were affected. Defecation was extremely painful because of the highly inflamed rectum which was raw and bled easily. Their temperatures were normal and appetites good. Two animals died after exhibiting symptoms of depression. All symptoms disappeared and no more animals were stricken after they were taken off the pasture containing the mushrooms.

Later an attempt was made, by Dr. Paul L. Rierly of the Louisiana State University, to poison a calf and a rabbit by gathering mushrooms and feeding them. Neither animal would eat the mushrooms voluntarily and they were force fed. The calf was not affected, probably because it was not fed a sufficient quantity to poison it. The rabbit was nauseated after about four hours but showed no other symptoms until death which occurred in 48 hours. Necropsy revealed gastroenteritis, red urine, dark-colored liver and ecchymotic hemorrhages on the heart.

H. A. BURTON

Alexandria, La.

Atypical Bladder Calculi of a Bitch

October 28, 1943, a white and brown Pekingese bitch, four years of age, weighing 5kg and in rather an emaciated condition, was presented for treatment.

Anamnesis.—The owner complained that his dog had been having difficulty in micturating; she dribbled quite frequently and displayed a great deal of pain with each effort. He had first noticed these symptoms about a year previously while living in Colorado Springs. The dog was immediately taken to a small animal clinician and a very small calculus was removed by way of the urethra. This seemed to bring immediate relief. She appeared to do well for two or three months; when suddenly, she began to show the same symptoms again, although not so severe. However, they grew more severe until finally she was in such continual pain that it was almost impossible for her to move about normally.

The present owner raised the bitch from the time she was two weeks of age. He had started her on milk and bread. Later, she had been fed scraps from the table. From 1941 until the present date her feed consisted entirely of a commercial dog food.

Diagnosis.—In an X-ray photograph, taken through the pelvic region, there appeared to be a solid black mass occupying the entire bladder. The presence of such a mass was confirmed by manual palpation of the bladder through the abdominal wall. Urinary calculi were identified.

Prognosis.—Guarded.

Treatment.—The bitch was placed on the table in dorsal recumbency. The field of operation, just anterior to the pubis, was shaved and properly disinfected. Ether was administered. An incision was made approximately three inches in length so that the bladder was well exposed. Small hemorrhages were controlled by hemostats. The bladder was sufficiently wrapped with gauze to prevent urine from flowing into the abdominal cavity, and an incision was made through the walls where blood vessels show the fewest anastomoses. However, after incision it was found that the original opening was not large enough to allow the removal of the unusually large calculi, so it was enlarged. When the bladder was opened viscid, foul and cloudy urine escaped. Seven large calculi were removed with surgical forceps. A thorough search was made for smaller stones but none were found.

Above: The bitch and the calculi removed from her bladder. Below: "Close-up" of the calculi

A double row of catgut sutures (Lembert) were used to bring the serous coats in direct apposition. They were placed close together to prevent urine escaping while adhesions were forming. The laparotomy wound was sutured in the usual manner, urea powder was sprinkled upon the operative area and the wound carefully bandaged. The animal was immediately given 21 grains of sulfathiazole followed by 7.7 grains every four hours for three days. An uneventful recovery followed.

Discussion

Occurrence.—Urinary calculi (uroliths) are formed in the renal pelvis, ureters, urinary bladder and urethra. Calculi cause more disturbances in a dog than in any other small animal. According to some authorities the cause of urinary calculi is yet to be discovered, however, some of the following factors are believed to contribute a great deal to mal conditions:

1. Local disturbances in urinary organs. In such a case there will be found degenerative changes from products of the urine (uric acid) leading to necrosis of the cells. These products become the nuclei around which the salts are deposited to form the calculi.

2. Disturbances of metabolism in which there is a larger than usual quantity of material eliminated in the urine in the form of phosphates, carbonates and oxalates (calcium and ammonium oxalates). The excess of these salts is deposited around the organic nucleus, leading to a calculus.

It is assumed by some authorities that, perhaps, certain types of bacteria, such as *Escherichia coli*, may favor the formation of calculi. The bacterial invasion of the bladder is believed by some to be facilitated by a vitamin A deficiency. The protective colloids in the urine may flocculate under these conditions and aid in the cohesion of the precipitated salts, or bacteria themselves, together with the products of the inflammation caused by them, may form the nuclei of such calculi.

Chemical Composition.—Each species of domestic animal has its chemically characteristic uroliths. In the carnivora they usually consist of calcium carbonate, phosphate and urate, sodium and ammonium urate or ammonium-magnesium phosphate.

Pathogenesis.—Urinary calculi are dangerous, chiefly because they may incite an inflammatory reaction and may occlude the urinary passages. This may result in grave conditions such as retention of the urine with its accompanying uremia, dilation and rupture of the bladder if the urethra be involved, or conversion of the kidney into a thin-walled sac (hydronephrosis) if the ureter be obstructed.

Symptoms.—The acid concretions do not, as a rule, produce any marked symptoms, except as they impede the flow of the urine.

The large alkaline stones cause most of the predominant lesions of clinical vesical lithiasis. Usually they are the ones that require radical treatment. There will be catarrhal inflammation of the bladder and pus cells in the urine. Micturation will be painful with only small quantities of urine passed. Constant dribbling of the urine is the most common symptom.

Examination of the bladder is necessary to determine the presence of calculi. This may be accomplished either digitally through the rectum or by abdominal palpation or laparotomy.

Prognosis.—When cases are taken early, before systemic disturbances from absorption of urine, etc., occur, the prognosis is usually favorable. However, the local disturbance in the bladder produced by the calculi should be taken into consideration, for sometimes serious lesions, difficult to heal, may be found in the mucosa.

HARRY E. CHILES.

Topeka, Kans.

Comminuted Fracture of the Mandible in a Dog

A brown mongrel (poodle-type) male, 1½ years of age, was presented for treatment August 22, 1943, two days following an injury

Figure 1

received in an automobile accident. The obvious injury was a comminuted compound fracture of the mandible. The fracture (Fig. 1) was just posterior to each canine and exposed

the root of one tooth. Numerous cinder particles were embedded in the fracture area with consequent soft tissue necrosis and involvement of the fractured ends of the bones.

Systemically, the dog showed marked depression and dyspnea. The temperature was 102°F. and the pulse accelerated. The erythrocyte count was 2.4 million and the leucocyte count 29,200. Sixty cubic centimeters of whole blood (anticoagulant heparin) was given intravenously and 25% glucose *per os* as required.

Nembutal anesthesia was administered and the fractured area exposed. Débridement was necessary for the soft tissue and the fractured ends were curretted. The wound was irrigated with a 1 to 2000 solution of tyrothricin. An aluminum plate was shaped to the contour of the mandible extending from the incisors to a point beneath the last molar. Holes were drilled in the plate to coincide with the interdental spaces of the premolars 2nd and 3rd, 3rd and 4th, 4th premolar and 1st molar, and 1st and 2nd molars. In addition two holes were drilled in the floor of the incisor portion of the mandible and two more just anterior to these. No. 20 gauge stainless steel wire was used in fixing the plate to the mandible. At this point the mandibular area was clipped and thoroughly washed with soap and water. A leather pad was used to cushion the attachment of plate to mandible. Using a straight Mayo intestinal needle the wire was passed from the plate dorsally to a point lingually and opposite an interdental space. The wire was then passed through the interdental space buccally and then ventrally through a hole in the plate. When all wires were passed there were four on either side posteriorally and two fixing the anterior fractured portion. Apposition of the fractured ends was then possible. The wires were knotted snugly, completing the immobilization.

Figure 2

Following a blood transfusion the erythrocyte count returned to five million while the leucocyte count gradually fell from 27,500 to 5,000 in eight days. No chemotherapy was employed other than irrigation with the tyrothricin solution. A fluid diet was given the first three days and then a semi-solid diet for the remainder of the convalescence.

Three weeks after it was applied the plate was removed as sufficient healing to maintain normal mandibular contour was evident by that time. However, at certain points in the healing area it was possible to remove small necrotic bone fragments. In two days a small swelling developed below the fracture. Drainage was established and a small amount of purulent fluid expressed. On probing a sequestrum was located and removed. After irrigation with 1:2000 tyrothrycin solution recovery was uneventful (Fig. 2) being complete about five weeks subsequent to the accident.

R. L. BURKHART

New Brunswick, N. J.

Choosing a Location for Practice

One of the foremost problems in a senior veterinary student's mind just before graduation is, What shall I do when I graduate? I know that was my problem and the problem of many others in our graduating class.

Many go into government work, some have a member of their immediate family in veterinary service with whom they can associate. However, many less fortunate have to choose a location.

My advice based upon my own experience is to choose a small city having a sustaining industry with a considerable amount of agriculture in the surrounding country. In such a locality, a considerable number of families have a family cow or two which constitutes a regular source of income to the practitioner. There are also hunting enthusiasts and dog breeders whose pointers, setters, hounds, retrievers, etc., require our services. And, as elsewhere, there are always the pet dogs and cats which need our ministrations every so often.

In my opinion, the first citizen to contact when locating in a community is the county health officer or local health department official. One can inquire as to the extent of the meat and milk inspection of the town. If there are no provisions made for either, the health officer may soon devise some plans for having this work done and that will afford an opportunity for the veterinarian. In my own case

it resulted in an immediate appointment to the position of city meat inspector and supplied a modest but regular income and, perhaps, what is more important, this served as a means for getting acquainted.

Another source of income that will soon develop is the testing of cattle for tuberculosis and brucellosis. Testing cattle also offers a fine opportunity to meet another group of citizens —farmers, dairymen and stock raisers of the locality. This official cattle testing is work that the empirics can't touch.

Practically all the empirics in my community are old men who are gradually passing out of the picture. Most of them have taken, at some time, a correspondence course in veterinary medicine and have picked up a few prescriptions and "receipts" for the different ailments. Since they were already in business when the veterinary practice law was enacted, they were given a permit to continue practice. Some of them are fairly competent. We have one empiric here who has practiced for 35 years and another who has had 40 years experience. Quite a bit of knowledge can be

Choose a small city with considerable agriculture in the surrounding country

picked while dealing with livestock for that length of time.

My particular locality at one time was the largest yellow pine lumber center in the world and large sawmills were located here. Quite a number of horses, mules and oxen were kept in the woods and at the mills. One of the empirics looked after all the horses and mules for the mills and gleaned quite a bit of useful information from his experience. After the mills were moved he went into general practice, where he sometimes gets into deep water.

In the 4½ years that I have been practicing in Laurel, I have encountered these empirics pretty often. However, they don't hurt my practice and frequently help me by turning calls my way, admitting that "they are not up on these new-fangled treatments and remedies" which produce quicker and surer results than the old empiric remedies.

If the prospective veterinarian is looking for a location for general practice, he can invariably get his wish in a place of this type. There are numerous such locations.

We never know what to expect in such a varied practice. We may be called to treat a cow with milk fever, then a dog with a fractured leg, a mule with spasmodic colic, or to deliver a calf or a litter of puppies, all during one day of practice.

As a rule, collections are pretty good in a small community. The good veterinarian is considered a valuable asset to his community and is treated as such.

I haven't mentioned the County Agent. One should call upon him early. If he is inclined to be co-operative he can be very helpful to a new veterinarian. The veterinarian can and should be helpful to him also. If he is not inclined to be co-operative, it is unfortunate, but that does not justify one in starting a fight with him or speaking disparagingly about him. There are obstacles enough in starting a new practice without developing unnecessary ones.

Therefore, I believe, if a senior student is looking for a location, he could not go wrong by choosing a location in a small community possessing at least one thriving industry which affords regular employment to a considerable number of the citizens.

H. B. TITLE

Laurel, Miss.

Dog Survives 24 Days' Fast

Mr. M. C. Green, a client of mine who owns 25 hunting dogs, was hunting with some neighbors 16 miles north of the city one night. One of his pack of hounds "Nelson Eddy" disappeared and although a search was made he could not be located. As he did not return home the next day, on the second day of his absence, Mr. Green made a systematic search of the countryside and notified all residents of his loss.

Just 24 days after his former hunt, Mr. Green, with the same party, was again hunting in the same area. His attention was attracted by a faint howl and investigation discovered Nelson Eddy at the bottom of a dry, abandoned well. Except for having lost between 30 and 40 pounds of weight, the hound was in good condition. Mr. Green has hunted for 65 years but says the greatest thrill he has ever experienced on a hunt was seeing Nelson Eddy coming out of that pit. It is reasonably certain the dog had no food during his imprisonment and doubtful that he was able to get any appreciable amount of water from such rainfall as fell into the well.

DON KITCHEN

Greenville, S. C.

Book Reviews

Taber's Cyclopedic Medical Dictionary including a Digest of Medical Subjects—Medicine, Surgery, Nursing, Dietetics and Physical Therapy—by *Clarence Wilbur Taber* and 14 associates. Second Edition, 1488 pages, 273 illustrations. Published by F. A. Davis Company, Philadelphia, 1944. Price $3.00.

The first edition of this work proved quite popular with veterinarians. The new edition, which has been entirely rewritten, contains more than twice as many words and yet sells at the same low price as the former edition.

This work is a dictionary of medical subjects as well as of medical terms—a medical encyclopedia and medical lexicon in one. Features claimed for it are:

It contains every important medical word—more than 50,000 medical terms.

Capitalization is indicated for all words.

More than 99% of the vocabulary words are respelled for pronunciation.

The derivation of practically all vocabulary words is given.

The definitions are complete, concise and set off in a separate paragraph from the additional information which is given. With important words a collateral list, containing all words pertaining to the same subject, is given.

Virus Diseases in Man, Animal and Plant. By *Gustav Seiffert*. Translated by *Marion Lee Taylor*, Ph.D. 332 pages. Published by Philosophical Library, New York, 1944. Price $5.00.

This work, a survey and reports covering the major research work done on viruses during the last decade, is stated to have been published upon the recommendation of the National Research Council. The description of diseases of plants comprises only a dozen pages but all important virus diseases of man and animals are discussed, many quite briefly.

The literature about viruses and virus diseases has increased greatly in recent years and the author finds the virus question so much in a state of flux, uncertain and unclarified, that even the significance of publications cannot always be judged correctly. He poses more questions than he answers.

The illogically coined word "vira" is used with annoying frequency and, as if that were not distressing enough, the author states that "the word 'vira' is today generally used for the plural [of virus]." Of course such is not the case.

The discussion of the virus diseases of animals is quite comprehensive—all the important ones at least receiving consideration. The failure to mention the Kelser vaccine for the prevention of rinderpest, although older and less effective measures are discussed, is puzzling. The discussion of hog cholera contains a number of errors and neither crystal violet nor tissue vaccine are mentioned.

What Dog Is That? By *Walter Edward Blythe* with descriptive text by *Joseph Nathan Kane*. 60 pages, 122 illustrations. Published by Greenburg, New York, 1944. Price $1.00.

This is an identification manual for all breeds of dogs recognized by the American Kennel Club. The illustrations, which includes the head of an animal typical for each breed, were drawn by an artist whose hobby is dogs. The descriptive matter concerning each breed (50 to 100 words) is condensed and gives history, uses, type of coat, colors, shape of head, height and weight and other description deemed pertinent by the author.

The object of this volume is to enable one to classify as to breed, any dog that he sees.

Any book reviewed in these pages, or any other book if available, may be obtained by remitting the published price to VETERINARY MEDICINE, 7632 S. Crandon Ave., Chicago 49. Bulletins and Circulars should be requested direct from the address given in the notice. Generally they are supplied free unless a price is stated.

The New Chemical Formulary VI. By a board of 75 editors; *H. Bennett*, F.A.I.C., Editor in Chief. 636 pages. Chemical Publishing Company, Inc., 1943. Price $6.00.

The publisher states that this work "contains thousands of the latest tested formulae from every important industry." The reviewer doubts that even the Editor in Chief knows how many formulae this book contains. "Thousands" seems a conservative enumeration. The formulae include those for adhesives, beverages, cosmetics, drugs, emulsions, farm and garden specialties; food products, hides, leather and fur, inks and marking materials; lubricants and oils, construction materials, metals alloys and their treatment; paint, varnish, lacquer and other coatings; paper, photography, polishes and abrasives; pyrotechnics and explosives, rubber, resins, plastics and waxes; soaps and cleaners, textiles and fibers and hundreds of miscellaneous, such as formulae for making artificial fish bait, artificial teeth, mustache and eyes; finger print powder and a thief detector; for making small animals transparent, meerschaum, yeast nutrient, and a feather preservative. It almost seems one would be safe in offering a prize for the name of anything for which the formula is not given.

A distinctive feature is a list of manufacturers from whom the materials named in the formulas may be obtained.